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AMNIONVOG-EMBOLISME (AMNIOTIC FLUID EMBOLISM)

'N BEWESE GEVAL EN 'N KORT SAMEVATTING VAN DIE LITERATUUR

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Daar is nou reeds soveel gevalle van amnionvog-embolisme gerapporteer wat deeglik gestaaf is, dat die bestaan van hierdie seldsame dog katastrofiese verloskundige komplikasie as 'n patologiese entiteit nie langer betwyfel kan word nie.

Meyer[1] (1926) was die eerste om die toestand te beskryf, maar dit was eers in 1941 dat Steiner en Lushbaugh[2] die kliniese implikasies ondersoek, bevestig, en eksperimenteel bewys het nadat hulle 8 gevalle wat skielik in kraam met skok oorlede is, nagespeur het. Baie van hierdie gevalle het voorheen waarskynlik te lande gekom in die kategorie van sogenaamde ‚idiopatiese obstetriese skok', postpartum bloeding, idiopatiese longedeem, ens.

BESKRYWING VAN 'N GEVAL

'n Kleurlingvrou, 29 jaar oud, para 7, is as 'n noodtoelating opgeneem by 35 weke swangerskap met 'n geskiedenis van 'n pynlose vaginale bloeding van ± 6 onse, 3 dae vantevore en sedertdien 'n aanhoudende, effens bloederige vaginale afskeiding. Voor die bloeding was die swangerskap normaal. Die vorige swangerskappe en verlossings was sonder voorval.

By Ondersoek

Buik 35 weke grootte, verteksligging, skedel in die bekkeningang in die L.O.A.-posisie, en die fetale hart gereeld teen 'n snelheid van 140 per minuut. Geen teerheid of buikspanning teenwoordig nie.

Algemeen. Effe edeem. Hemoglobien 10·5 g. %. Pols 88 per min. Bloeddruk 165/110 mm. Hg. Die urine was vry van eiwit en suiker. Geen vaginale bloeding. Bloedgroep O, Rh. positief, stollingstyd 3 minute, ‚Fibrindex'-toets: 5 sekondes.

Vaginale ondersoek. 'n Vae massa is in die anterior forniks gevoel. Die os was 2 vingers ontsluit. Plasenta previa, graad I-II gevoel. Geen bloeding. Vliese nie gebreek nie weens prematuriteit.

Behandeling. Soutvry dieet, reserpien 0·25 mg. t.d.s., chlorotiasied 0·5 g. b.d. en puroverin retard 1 tablet elke 4 uur.

Na Toelating

Een dag na toelating. Toestand bevredigend, bloeddruk 130/90 mm. Hg.

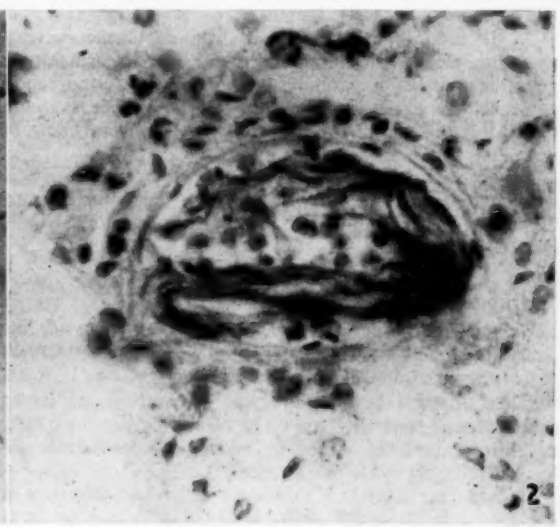
Drie dae na toelating. Pols 120 per minuut, temperatuur 99·6° F. Fetale hart nie hoorbaar nie, sametrekkings waarneembaar en 'n onwelriekende vaginale afskeiding teenwoordig. Pasiënt sweet baie en daar is 'n effense hoes teenwoordig. Longvelde skoon. Vaginale depper geneem en penisillien en streptomisien toegedien.

Tien uur later is 'n manlike doodgebore baba, gewig 6 pd. 5 onse, spontaan verlos. Moederlike pols 140 per minuut, temperatuur 101° F. Na 'n derde stadium van 10 minute is die plasenta spontaan en volledig verlos, onmiddellik gevolg deur 'n hewige, massiewe bloeding uit 'n verslapte uterus. Binneaarse metergin 0·4 mg., is gegee en 'n pitosien-indruppeling en bloedoortapping is begin. Uterus trek beter saam en bloeding verminder, maar pasiënt se pols en bloeddruk nie waarneembaar nie en terwyl tweede pint bloed ingepomp word, sterf die pasiënt. Totale bloedverlies: ±100 onse.

Nekropsie

Algemene ondersoek van die liggaam het geen positiewe bevindinge opgelewer nie, behalwe vir bleekheid van die naels

Afb. 1, ×450. Fibrienkleuring dui aan twee klein pulmonale bloedvate met genukliëerde plaveiselepiteelselle teenwoordig in die lumina.



Afb. 2, ×450. Fibrienkleuring dui aan 'n pulmonale arteriool met musienagtige debris tesame met 'n paar rooibloedselle teenwoordig in die lumen van die bloedvat.

lippe en konjunktivas. Die lykskouing is gedoen 27 uur nadat die pasiënt oorlede is.

Die hart was van normale grootte, uitgeset en sag, met effense bleekheid van die miokardium. Die regter-ventrikel, pulmonale arterie, asook die vertakkings daarvan, het vloeibare bloed bevat. Die longe het 'n geringe atelektatiese voorkoms gehad met donker, hiperemiese, vogtige sny-oppervlaktes, terwyl die longbloedvate makroskopies geen merkwaardige tekens van belang getoon het nie.

Die lewer was effens vergroot, sag en bleek, terwyl die milt 'n normale grootte gehad het met 'n gerimpelde kapsel en hiperemiese pulpa. Die byniere en pankreas was makroskopies normaal. Die niere het 'n effense granulêre, bleek korteks getoon, waarvan die kapsels taamlik moeilik afgestroop het, terwyl die niermedullas 'n meer hiperemiese voorkoms gehad het.

Die uterus was ongeveer die grootte van 'n swangerskap van 30 weke en het by ondersoek geen patologiese veranderinge getoon nie. Die fetus was by ondersoek 'n manlike, doodgebore baba van 2,854 g. met die algemene tekens van outolise teenwoordig, terwyl die plasenta ook niks merkwaardigs van belang aangetoon het nie.

Daar is oorspronklik gemeen dat die oorsaak van dood skok was as gevolg van postpartum bloeding en gevolglik is daar gesoek na intravaskulêre deponering van fibrien. Daar is dus voortgegaan met fibrienkleurings, naamlik Weigert se metielviolet-fibrienkleuring en die fosfotungstaansuur, hematoksilien- en eosienkleuring, op snitte van die longe, hartspier, lewer, niere en uterus.

Daar is by ondersoek van die longweefsel wat só gekleur is, opgemerk dat musienagtige debris, epiteelselle, asook lanugo teenwoordig was in die kleiner pulmonale arteries en arteriole, op grond waarvan die diagnose van amnionvog-embolisme dus gemaak is.

Verdere histologiese bevindinge in die longe was dié van hiperemie met geringe pulmonale edeem terwyl die lewer troebel swelling van die lewerselle en geringe diffuse vetterige degeneratiewe veranderinge aangetoon het. Die nierbuisie-epiteelselle het matige vetterige degeneratiewe veranderinge aangetoon terwyl die kapsels van Bowman effens verdik was.

Histologiese ondersoek van die doodgebore baba het slegs hiperemie van die longe, lewer, milt en leptomeninges aangetoon.

Opsomming

Die kardinale punte van hierdie geval is dan: Plasenta previa Graad II anterior, met pre-eklampsie; doodgeboorte; amnionvog-embolisme en postpartum bloeding; en moederlike sterfte.

Kommentaar

As die moontlikheid van amnionvog-embolisme oorweeg was voor die verlossing, kon die nodige voorsorgmaatreëls getref geword het, nl. stollingstoetse, bloed en fibrinogeen in gereedheid, ergometrien binneaars na die tweede stadium, ens. Moontlik kon die moederlike sterfte dan vermy geword het.

In nabetragting, na die lykskouing, was dit duidelik dat die amnionvog-embolisme waarskynlik begin het voor die verlossing op dié stadium toe die pasiënt polsversnelling, koors, ens. begin ontwikkel het.

BESPREKING

Voorkoms

Die toestand is seldsaam, maar met groter bewuswording daarvan sal stellig 'n hoër voorkoms gevind word. Steiner en Lushbaugh[2] het die voorkoms bereken op 1 : 8,000 bevallings, terwyl Barno en Freeman[3] gedurende die ‚Minnesota maternal mortality study' 'n voorkoms van 1 : 37,000 bevallings gevind het. As 'n oorsaak van moederlike mortaliteit het hulle dit vyfde in volgorde gevind!

Hoewel daar al verskeie gevalle met herstel gerapporteer is, bv. Firth,[4] is dit moeilik om hierdie gevalle as sodanig te aanvaar aangesien die finale diagnose hier noodwendig op nadoodse histologie berus. Sover bekend is die geval wat hier gerapporteer word die eerste in die Suid-Afrikaanse literatuur.

Kliniese Beeld

(a) Simptome en Tekens

Die uitstaande, feitlik kenmerkende beeld is skielike skok, tagikardie, rusteloosheid, dispnee en sianose, soms voorafgegaan deur kouekoors of braking. Dit kan gedurende, net voor, of net na die verlossing plaasvind. Dood volg gewoonlik onmiddellik of binne 'n paar uur. In ±40% van pasiënte wat die onmiddellike insident oorleef, ontwikkel 'n stollingsdefek wat dan ook weer kan lei tot sterfte as gevolg van 'n massiewe postpartum bloeding. Ander bevindings wat soms teenwoordig is, is pulmonêre edeem, akute cor pulmonale, regter ventrikulêre versaking, en hiperpireksie. Pyn in die borskas of epigastrium kom ook soms voor, maar is gewoonlik nie 'n uitstaande simptoom nie. Röntgenologies is die beeld soos dié van miliêre longtuberkulose.[32]

(b) Aanduidende Omstandighede en Predisponerende Faktore

'n Kombinasie van die volgende faktore word gewoonlik in wissellende verhouding gevind: stormagtige, tetaniese uteruswerking, hoë pariteit, hoë ouderdom, groot baba, pitosiengebruik, gebreekte vliese, mekonium in die vrugwater, intra-uterine dood, en verlengde swangerskap. Die mees konstante faktore is hewige sametrekkings van die uterus en gebreekte vrugvliese.[14]

(c) Ander Bevindings

1. In die gevalle wat die onmiddellike episode oorleef, maak bloedingsneigings weens 'n versteurde stollingsmeganisme dikwels hul verskyning. Gewoonlik is daar 'n demonstreerbare verlaging van al die stollingskomponente van plasma—fibrinogeen, protrombien, plaatjies en die versnellingsfaktore soos Faktor V en Faktor VII. Die klem val egter op die fibrinogeenverlaging omdat die toestand gewoonlik gekorrigeer kan word deur fibrinogeen alleen toe te dien en omdat die vermindering van die ander plasmabestanddele gewoonlik nie ernstige afmetings aanneem nie.[12]

Soms is daar ook heparienagtige stowwe[7,8] teenwoordig in die bloed, met of sonder fibrinogeenverlaging, wat die bloedingsneiging veroorsaak. In amnionvog-embolisme speel hierdie proses waarskynlik 'n groter rol as in die ander tipes van die ‚obstetriese bloedingsindroom'[20] soos abruptio placentae, teruggehoue dooie fetus, septiese aborsie, ens.

Aktiewe fibrinolisiene[9-10,20] is ook al herhaaldelik gedemonstreer in gevalle wat met abnormale bloeding voorgedoen het.

Behalwe die meer ingewikkelde en tydrowende laboratoriumbepalings kan die volgende toetse onmiddellik by die bed uitgevoer word om die aard van die defek te help bepaal:[30]

(i) Wiener se stolselwaarnemingstoets.

(ii) ‚Fibrindex'-toets (trombien+plasma van pasiënt+kalsium).

(iii) Vir heparien: pasiënt se bloed+‚normale bloed'=geen stolsel.

(iv) Vir fibrinolise: pasiënt se bloed+gestolde ‚normale bloed'=stolsel los op.

2. In gevalle wat sterf en waar 'n lykskouing nie gedoen kan word nie, kan volgens Gross en Benz[15] se aanbeveling 'n naald en spuit gebruik word om regter-ventrikelbloed af te trek en hierin dan te soek na vrugwaterbestanddele.

3. Aangesien hartkateterisasie vandag as 'n betreklike vereenvoudigde en veilige prosedure uitgevoer kan word, is dit deur Barno en Freeman[3] voorgestel as 'n moontlike metode om 'n monster bloed vir ontleding te bekom in ver-

dagte gevalle. Dit sal egter maar selde 'n praktiese uitvoerbare hulpmiddel tot diagnose wees.

Om die tipiese beeld te illustreer kan die volgende geval[3] as voorbeeld geneem word: Die pasiënt, 24 jaar oud, para 1, is toegelaat 3 weke na haar verwagte datum vir 'n elektiewe kraaminduksie. Voorgeboortelik was alles normaal. Pitosien-indruppeling, 6 eenhede per 100 ml. om die serviks ‚ryp' te maak, is 3 maal herhaal. Een uur later het sy koue rillings gekry—temperatuur 102°F. 'n Amniotomie is gedoen en 'n pitosieninfuus herhaal. Geen aktiewe baring nie. Volgende dag: pitosien is herhaal waarop hewige sametrekkings gevolg het nadat die spoed van die indruppeling vermeerder is tot 48 druppels per minuut. Ses uur later, by volle ontsluiting van die serviks, het die pasiënt skielik kortasem, sianoties, pols- en bloeddrukloos geword, en 'n stuiptrekking ontwikkel. Fetale hartklanke nie meer waarneembaar nie. Sterfte het onmiddellik daarna gevolg.

Nekropsie. Bloed in die regter-atrium bevat sigbare lanugo en mikroskopies is die longbeeld tipies van amnionvog-embolisme. Verdere bevindings almal negatief.

Differensiële Diagnose

Veral twee groepe toestande moet onderskei word, naamlik gevalle met akute skok, wat soms oënskynlik ‚onverklaard' mag wees, asook ander oorsake van bloeding om en by die verlossing.

Behalwe toestande van 'n meer interne geneeskundige aard soos miokardiale infarksie, inwendige bloeding, longembolus vanweë veneuse trombi of lug, ens., moet veral abruptio placentae, uterusskeur, atoniese postpartum bloeding, akute inversie van die uterus, akute byniersversaking, eklampsie, Mendelson se suur-aspirasie-sindroom, anafilaktoïede skok van diverse oorsprong, onverenigbare bloedreaksies[22] en septisemie,[23] onderskei word. 'n Belangrike punt mag wees dat skok die bloeding voorafgaan.[2]

Patologiese Anatomie

Makroskopies is daar dikwels longedeem en die ander algemene letsels van dood in akute skok, soos bv. uitsetting van die hart.

Mikroskopies is die kardinale kenmerk die teenwoordigheid van amorfe debris, lanugo, musien, vet en gal, epiteelselle van fetusoorsprong, en, minder algemeen, fibrien-embolieë in die kleiner longarterioles en kapillêres.[2,11] Shotton en Taylor[17] se geval het ook vrugwaterbestanddele in die brein getoon.

Dit is reeds lank bekend dat chorionselle soms by nekropsie op pasiënte wat aan ander oorsake oorlede is in die puerperium, gevind word in die kleiner longvate.[12] Die teenwoordigheid van amniotiese debris word egter nie postmortem gevind in pasiënte wat toevallig aan ander oorsake oorlede is kort na kraam nie.

Deur rooiselle, gemerk met radio-aktiewe chroom, in die vrugwater te spuit en na die verlossing daarvoor in die moederlike sirkulasie te soek, het Sparr en Pritchard[13] gevind dat die infuus van amnionvog gedurende normale baring nie plaasvind nie, d.w.s. die teenwoordigheid van amniotiese debris in die longvate is patologies.

Volgens onlangse werk van Attwood[11] kan met spesiale kleuringsmetodes (‚Alcian green' vir musien en ‚phloxin' vir epiteelselle) emboliese debris duideliker en in groter hoeveelheid aangetoon word as met hematoksilien en eosien. Hierdie metode toon soms embolieë wat nie met die gewone kleuring gevind word nie en word tans uitgevoer op die weefsels van die pasiënt.

Gross en Benz[15] het dit ook in die drywende deel van regterventrikel bloed met afswaaiing gevind. Drie lae vorm, en van die boonste laag kan smere gemaak en gekleur word om vrugwaterbestanddele aan te toon.

Patogenese

Dat amnionvog, veral as dit met mekonium bevlek is, by binneaarse indringing onmiddellike dood, hewige skok en bloedingsneigings kan veroorsaak[2,7] asook dat dit tromboplastienaktiwiteit bevat,[5,16] is herhaaldelik deur verskeie werkers eksperimenteel bewys by diere, bv. Steiner en Lushbaugh,[2] Reid, Weiner en Robey,[5] en Schneider.[7]

Klinies kan hierdie proses slegs plaasvind as die vliese hoër op geskeur is en nog heel is oor die interne os, of as die vliese oor die serviks geskeur is, terwyl die fetus se voorliggende deel besig is om die serviks dig af te sluit. Vermoedelik kan die vrugwater dan intravaskulêr toegang tot die moederlike sirkulasie vind, eerstens deur middel van die veneuse sinusse onder die plasenta as daar gedeeltelike loslating is; tweedens, deur middel van die servikale venes as daar klein skeurtjies teenwoordig is; en derdens, tydens blootlegging van die moederlike venes in die uterus, wanneer die uterus skeur, of by keisersnit of plasenta previa.

Volgens Steiner en Lushbaugh[2] is die meganisme van dood hoofsaaklik meganiese embolisasie van die pulmonêre sirkulasie deur die amorfe deeltjies, gevolg deur pulmonêre arteriële kramp en 'n meegaande anafilaktoïede skokreaksie. Schneider[7] stem grootliks hiermee saam nadat hy die beeld gereproduseer het deur honde met mekonium in te spuit. Hy postuleer ook dat die longarteriële kramp weens vrystelling van serotonien[18] uit afgebreekte plaatjies kan ontstaan. Op hierdie basis kan die stollingsdefek voorkom as gevolg van die vrystelling van heparienagtige stowwe, of weens die aktivering van fibrinolisien. Dit is welbekend dat laasgenoemde twee verskynsels kan plaasvind in enige toestand van ernstige skok.[7,19,21]

In anafilaktoïede skok lei die vasomotoriese kollaps tot heparienvrystelling as gevolg van vagusprikkeling.[20] Laasgenoemde teorie pas dan ook in by die gevalle van amnionvog-embolisme wat nie tekens van inkorting van die pulmonêre sirkulasie, of vermindering van die plasma-stollingskomponente toon nie.[7,21] Quick[22] stem saam dat heparienvrylating 'n rol mag speel, maar meen dat fibrinolise 'n toevallige bevinding is.

Hierteenoor staan die teorie van Reid, Weiner, en Robey[5] dat die skok en bloedingsneigings hoofsaaklik ontstaan weens tromboplastieninfuus met gevolglike wydverspreide intravaskulêre stolling (en defibrinasie met vermindering van al die stollingskomponente), en fibrienembolieë. Verskeie gevalle, wat hierdie teorie gedeeltelik staaf, is reeds gerapporteer.[23] Die feit dat daar min tekens van intravaskulêre fibrienedeponering by nekropsie gevind word, kan verklaar word deur die aktivering van die fibrinolisesisteem, soos beskryf deur Moloney *et al.*,[10] Astrup,[19] en andere.

McKay, Hertig, *et al.*[24] het ook op die patologiese ooreenkoms van amnionvog-embolisme (en ander akute noodlottige swangerskapstoestande), en die algemene Shwartzmanreaksie, waar fibrienembolise prominent is, gewys; ook dat in swangerskap, sowel as gedurende kortisoonbehandeling, slegs 'n enkele dosis ‚toksien' die reaksie kan presipiteer.

In Ratnoff en Vosburgh[6] se geval is bewys gelewer van hipofibrinogenemie, trombositopenie, antitrombienaktiwiteit, en fibrinolise.

By monde van Stefanini *et al.*[25] kan gesê word dat al die bogenoemde faktore waarskynlik 'n rol speel in 'n wisselende graad van belangrikheid van geval tot geval.

Behandeling

Hoewel die toestand feitlik altyd noodlottig is, sal die mortaliteit moontlik verlaag kan word deur wyer bewuswording van die entiteit. As voorbehoedende maatreël kan die onversigtige gebruik van pitosien sonder behoorlike toesig, beklemtoon word en in die geval van postpartum bloeding, waar die gewone oorsake uitgeskakel is, moet aan stollingsdefekte gedink word. Suurstof, morfien, binneaarse atropien en papaverien (om die vaguseffekte te blokkeer en die pulmonêre vate en brongiole te verslap), en aminofillien vir dieselfde doel, mag van waarde wees. Adrenalien is verbode weens die reeds bestaande arteriolêre kramp. Binneaarse vog en bloed moet weerhou word in die teenwoordigheid van akute pulmonêre edeem.

In gevalle wat met postpartum bloeding voordoen, is die eerste oorweging die vervanging van die bloedvolumeverlies, verkieslik met vars bloed wat al die stollingsfaktore soos Faktor V en Faktor VII, ook bevat. Bloedoortapping alleen kan egter nie altyd die stollingsdefek korrigeer nie en dan moet fibrinogeen gegee word.[26,31] Van 4 tot 12 g. mag nodig wees.[27] Binneaarse kortisoon, 100-200 mg., word gegee teen fibrinolise.[28] Protamiensulfaat, 20-50 mg., kan stadig intraveneus toegedien word as heparienagtige stof gevind is.[30] Dit moet egter baie versigtig toegedien word tensy dit gekontroleer kan word met titrasie, want 'n oormaat kan in sigself antistollend wees.[29]

Daar moet egter onthou word dat die tonus van die uterus, selfs in gevalle met 'n stollingsdefek, nog altyd die belangrikste hemostatiese meganisme is.[31] Gevolglik moet oksitosiese middels ook hier 'n belangrike plek in die behandeling van die bloeding inneem.

SUMMARY

A fatal case of amniotic fluid embolism in a Coloured multipara with toxaemia and placenta praevia, which resulted in an intra-uterine death and postpartum bleeding, is reported.

This is presumably the first case with histological evidence to be reported in the South African medical literature.

The condition is outlined and the relevant literature briefly reviewed.

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FORTHCOMING INTERNATIONAL MEDICAL CONFERENCES

The Fourth International Goitre Conference (second announcement), sponsored jointly by the American Goiter Association and the London Thyroid Club, will take place at the Royal College of Surgeons, Lincoln's Inn Fields, London, W.C.2, on 5-8 July 1960. Intending participants are asked to register as soon as possible, but not later than 1 June 1960. Those who wish to attend any single session only will be able to arrange this by Day-Registration at the time of the Conference. Members who register before 31 December 1959 will be required to pay a fee of £4 upon registration. For members who register after that date the fee will be £5. A numbered membership card will be issued when registration is completed. The fee for Day-Registration will be £2.

Participants are reminded that they must make their own travel and hotel arrangements. It is essential that reservations be made early, and may be done through Messrs. Thomas Cook & Son Ltd.

A fund is available to assist a limited number of scientists from abroad. Since the amount available for this purpose is small in proportion to total needs, all who can secure travel aid from other sources are urged to do so. Information on travel grants may be obtained from Dr. Selwyn Taylor, 3 Roedean Crescent, London, S.W.15. No application will be considered after 30 April 1960.

The programme will provisionally include 6 sessions on: Factors affecting thyroid function, hypothyroidism (2), endemic goitre (epidemiology and public health), hyperthyroidism, and cancer of the thyroid. There will be demonstrations and a full social programme has also been arranged. For information on papers to be considered for presentation see page 660 of the issue of this *Journal* for 8 August 1959.

The Nutrition Society of Great Britain. The 131st meeting of the Nutrition Society will be held in London on 19 March 1960, and will be in the form of a symposium on 'Nutrition and the elderly'. Information on the provisional programme may be obtained from the Hon. Programmes Secretary, D. F. Hollingsworth, c/o Ministry of Agriculture, Fisheries and Food, Great Westminster House, Herseferry Road, London, S.W. 1, or from Dr. B. Bronte-Stewart, Department of Medicine, University of Cape Town, Observatory, Cape.

The Second Afro-Asian Congress of Ophthalmology will be held in Tunis on 25-29 April 1960. During the meeting subjects such as trachoma, pterygium, etc. will be discussed. Ophthalmologists from South Africa will be welcomed at the Congress. Further information may be obtained from Docteur Ridha Mrad, Secretare General, 2nd Afro-Asian Congress of Ophthalmology, Centry d'Ophthalmologie, Tunis, N. Africa.

Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

VAN DIE REDAKSIE : EDITORIAL

VERGIFTIGING DEUR INSEKDODENDE MIDDELS

Daar is al herhaalde kere in hierdie *Tydskrif* gewys op die gevare van insekdodende en peswerende middels wat in die huis gebruik word sowel as deur die gesondheidsadministrasie, die landbou, die nywerhede, en die handel.[1-4] Baie internasionale besprekings oor vergiftiging met hierdie middels het al in verskillende lande plaasgevind, byvoorbeeld in Rome in 1953 onder beskerming van die W.G.O.

Gedurende Oktober het daar ook in hierdie land so 'n bespreking plaasgevind by geleentheid van die Jaarlikse Kongres van die Gesondheidsbeamptesvereniging van Suid-Afrika. Elders in hierdie uitgawe plaas ons 'n artikel deur prof. N. Sapeika[5] wat handel oor aspekte van die moderne insekdodende middels, hulle giftigheid, en beheer. Die algemene gebruik van hierdie gifsoorte maak dit noodsaaklik dat geneeshere en mediese studente op die hoogte van sake sal bly oor die gevare en die metodes van voorkoming en behandeling van vergiftiging van hierdie aard. Die komitee van die Amerikaanse Mediese Vereniging wat die probleem van peswerende middels ondersoek het, beveel aan dat hierdie middels en verwante produkte behandel moet word in die farmakologiekursusse van mediese skole.[6] Hierdie aanbeveling word gemaak ten spyte van die ongeëwenaarde aandrang op insluiting van nuwe onderwerpe in die farmakologiekursusse van die mediese leerplan.

Daar is 'n groot aantal chemiese stowwe wat as peswerende middels gebruik word en hul giftigheid verskil baie. Onder die middels wat op die oomblik taamlik algemeen gebruik word, is die chloor-(halogeen-) koolwaterstowwe en die organiese fosfaatesters. Die organiese fosfaatesters is veral baie giftig.

Die kliniese tekens van vroeë vergiftiging deur hierdie samestellings is nie spesifiek nie en dit mag moeilik wees om hulle te onderskei van gewone siektes. Die latere tekens het egter kenmerkende eienskappe. In sommige gevalle is daar sluimerende vergiftiging wat noukeurige mediese waarneming van die slagoffer nodig maak nog lank nadat hy oorspronklik aan die vergiftiging blootgestel was. In die geval van vergiftiging met dieldrin, byvoorbeeld, moet die pasiënt nog ten minste twee jaar lank na die oorspronklike behandeling en verwydering van die blootstelling, medies ondersoek word. Na vergiftiging met parathion moet die pasiënt ook lank onder observasie gehou word en bepalings van die terugkeer van cholienesterase-aktiwiteit in die serum tot die normale moet gemaak word—indien moontlik verskeie maande lank.

Die volgende interessante nuwe feite het as gevolg van navorsing gedurende die afgelope paar jaar aan die lig gekom. In verband met dikofaan (DDT), byvoorbeeld, is dit gevind dat herhaalde dosisse wat deur vrywilligers per mond geneem is oor 'n tydperk wat geduur het tot 18 maande, en teen ongeveer 200 keer die daaglikse sterkte waarin hierdie middel gewoonlik in voedsel voorkom, nie siekte veroorsaak nie. Dit dui op 'n groot veiligheidsfaktor ten opsigte van die inneem van DDT per mond. Die absorbering van groot hoeveelhede kan egter akute vergiftiging veroorsaak.

Met betrekking tot gamma bensien-hexachloried (BHC, gammexane) kan sensitiwiteit en gestelsreaksies ontstaan. Dieldrin, wat baie gebruik word in malaria-uitwissingsveldtogte, is 'n gevaarlike samestelling. Dit word deur die vel geabsorbeer selfs in die droë vorm. In hierdie opsig verskil dit dus aansienlik van DDT. 'n ‚Onwaarneembare' sweefmengsel oor die gesig, voorarms en ander velgebiede wat nie bedek is nie, is genoeg om vergiftiging te veroorsaak.[7] Ernstige siektetoestande soos epileptivorme aanvalle wat herhaaldelik kan voorkom en na vertraging kan ontstaan, en geestesversteurings, kan veroorsaak word. Dit is dringend noodsaaklik dat dieldrin verder bestudeer moet word, veral met die oog op berging in die liggaam, bepaling van die vlak daarvan in die bloed en in die urine, en die ontdekking van vroeë tekens van vergiftiging.

Wat betref die organiese fosfaatesters, soos byvoorbeeld, parathion,[4] kan ons meld dat daar heelwat navorsingswerk gedoen word, veral met betrekking tot die uitwerking daarvan op cholienesterase en die ontwikkeling van 'n siekteteenmiddel om die ensiem weer aktief te maak nadat dit onaktief geword het. In hierdie verband is wel al vordering gemaak deur die gebruik van piridien-2-aldoksien metiodied wat die muskarienagtige sowel as die nikotienagtige uitwerking van parathion teenwerk.[8,9] Atropien word in hierdie geval gelyktydig toegedien om die opeenhoping van asetielcholien op sekere plekke in die liggaam teen te werk. Parathion is baie giftig—'n enkel druppel in die oog kan noodlottige gevolge hê.[10] Hierdie gif word al meer gebruik as 'n middel om selfmoord mee te pleeg, soos byvoorbeeld gerapporteer in Finland en in Denemarke.[10] Dit is een van die giftigste middels wat gebruik word om pestoestande te beheer.

Gesien uit die chemiese, farmakologiese, kliniese en administratiewe aspekte, sowel as uit die oogpunt van moontlike vergiftiging deur hierdie middels, is daar baie interessante fasette verbonde aan die studie van kiemdodende en peswerende middels.

1. Aantekening (1952): S. Afr. T. Geneesk., **26**, 684.
2. Van die Redaksie (1953): *Ibid.*, **27**, 1149.
3. *Idem* (1954): *Ibid.*, **28**, 389.
4. Klugman, H. B. (1959): *Ibid.*, **33**, 899.
5. Sapeika, N. (1959): *Ibid.*, **33**, 1063.
6. Aantekening (1953): J. Amer. Med. Assoc., **152**, 709.
7. Hayes, W. J. (1959): Bull. Wld Hlth Org., **20**, 891.
8. Steyn, D. G. (1958): S. Afr. T. Geneesk., **32**, 894.
9. Aantekening (1959): Lancet, **2**, 166.
10. Toivonen, T. *et al.* (1959): *Ibid.*, **2**, 175.

INSECTICIDE POISONING

Attention has been drawn on a number of occasions in the *Journal* to the dangers associated with the use of insecticides and other pesticides used in the home, in health administration, agriculture, industry, and commerce.[1-4] Many discussions on poisoning with these agents have taken place in different countries and also at an international level, as for instance in Rome in 1953 under the suspices of the WHO.

The latest discussion in this country took place in Cape Town recently at the Annual Congress of the Health Officials' Association of Southern Africa. Elsewhere in this issue we publish an article by Prof. N. Sapeika[5] in which some details are given of modern insecticides and their toxicity and control. The widespread use of these poisons has made it necessary that physicians and medical students should be adequately informed about their dangers and the methods used for the prevention and treatment of insecticide poisoning. The Committee on Pesticides of the American Medical Association has urged the introduction of a study of pesticides and related products into the pharmacology courses of medical schools.[6] This is being done notwithstanding the unprecedented demand for including additional subjects in pharmacology in the medical curriculum.

A great variety of chemical agents have been used as pesticides, and their toxicity varies a good deal. Among those widely used at present are the chlorinated (halogenated) hydrocarbons and the organic phosphate esters, the latter being particularly toxic.

The clinical signs of early poisoning by these compounds are not specific and may be difficult to distinguish from the signs of an ordinary illness. The late signs have characteristic features. In certain instances there is a latent intoxication which necessitates close medical observation of the victim of poisoning for a long time after the original exposure. For example, in dieldrin poisoning the patient should be examined medically for at least two years after the initial treatment and removal from exposure. Also, after parathion poisoning prolonged observation and estimations of the return of serum cholinesterase activity to normal should be made, possibly for several months.

Some interesting newer facts that have emerged from studies in recent years may briefly be cited here. In experiments with dicophane (DDT), for instance, it has been found that repeated oral doses taken by volunteers for periods up to 18 months, at about 200 times the daily rate at which a man might receive this insecticide in his diet, does not produce illness, indicating a large safety factor as far as this mode of entry is concerned. Acute poisoning from the absorption of large amounts of DDT can, of course, produce serious symptoms.

With regard to gamma benzene hexachloride (BHC, gammexane), sensitivity and systemic effects can be acquired. Dieldrin, which has been much used, for example in malaira campaigns, is a dangerous compound. It is absorbed through the skin, even in the dry form, in this respect differing considerably from dicophane. An 'imperceptible' mist of the suspension on the unprotected face, lower arms, and other skin areas, is sufficient to cause poisoning.[7] Serious illness may be produced, including epileptiform seizures, which may be recurrent and delayed in onset, and mental disorder. There is urgent need for further study of dieldrin, its storage in the body, its estimation in blood and urine, and the detection of early signs of intoxiaction.

In the organic phosphate esters, for example parathion, much research work has been in progress especially with regard to its action on cholinesterase, and the development of a suitable antidote to reactivate the enzyme when it has been inactivated. In this connection an advance has been made with the introduction of pyridine-2-aldoxime methiodide which counteracts the muscarinic as well as the nicotinic effects of parathion,[8,9] atropine being given concurrently to counteract the accumulation of acetylcholine at certain sites in the body. Parathion is a very dangerous poison; a single drop in the eye may have a fatal effect.[10] It has been used to an increasing extent as a means of suicide, for example in Finland and Denmark.[10] It is one of the most toxic agents used for pest control.

There is much of interest and of practical importance in the field of insecticide poisoning from the chemical, pharmacological, toxicological, clinical, and administrative points of view.

1. Annotation (1952): S. Afr. Med. J., 26, 684.
2. Editorial (1953): *Ibid.*, 27, 1149.
3. *Idem* (1954): *Ibid.*, 28, 389.
4. Klugman, H. B. (1959): *Ibid.*, 33, 899.
5. Sapeika, N. (1959): *Ibid.*, 33, 1063.
6. Annotation (1953): J. Amer. Med. Assoc., 152, 709.
7. Hayes, W. J. (1959): Bull. Wld Hlth Org., 20, 891.
8. Steyn, D. G. (1958): S. Afr. Med. J., 32, 894.
9. Annotation (1959): Lancet, 2, 166.
10. Toivonen, T. *et al.* (1959): *Ibid.*, 2, 175.

MOTOR CAR INSURANCE

The arrangement which the Medical Association of South Africa has had with Messrs. Edward Lumley & Sons (Pty.) Ltd. for the last eight years has been terminated by the Company. It will be remembered that a reduced premium was available to members on policies issued by this Company when the rates on motor car insurance were generally increased. Edward Lumley & Sons have found that their claims experience has been such that it has become impossible for them to continue and after the end of 1959 they will not renew any policies which fall due for renewal.

We regret that this has become necessary for our relation with the Company have been pleasant and satisfactory and we have always found them cooperative. We shall also miss the commission which was paid to our Medical insurance Agency for this business.

After much thought, it has been decided that the interests of our members will best be served by advising them to place their motor car insurance, when it falls due, with the Atlas Assurance Company.

The Atlas is well known to our members, so many of whom are covered by the Company by Doctors' Professional Liability Insurance. The Atlas have been the official insurers to the Association for some thirty years and the relationship between the Association and the Company has always been happy. We know that those of our members who follow the advice of the Association's Insurance Agency

and place their motor car insurance with the Atlas will be satisfied with the treatment they receive. The premium rates will be based on the normal tariff rates, but the service they will receive will be that which we have come to expect of an organization of the quality of the Atlas Assurance Company.

SUBDURAL EFFUSIONS WITH RESPIRATORY INFECTIONS IN THE FIRST YEAR OF LIFE

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Department of Paediatrics, Karl Bremer Hospital, Bellville, Cape, and the University of Stellenbosch

Since the papers of McKay *et al.*[1,2] in 1950 and 1953 drawing attention to the occurrence of subdural effusions with meningitis, it has become commonplace to consider this complication when cases of meningitis, especially those due to *H. influenzae* and *S. pneumoniae*, fail to pursue their expected course with antibiotic treatment. Although the explanation for the occurrence of subdural effusion with meningitis remains obscure, at least the lesions are both intracranial and the complication therefore the more easily remembered.

Other diseases have been associated with subdural effusions,[3-5] especially inflammatory disease of the respiratory system and intestinal tract, and in some cases apparently simple malnutrition. Subdural effusions occurring at the time of birth from injury or haemorrhagic disease have long been known. Because there seems no immediate reason why infections of the respiratory and intestinal tracts should be found with subdural effusions, the association is therefore the less easily remembered, and so we report here 9 cases of respiratory infections in which this complication impeded recovery. The respiratory disease varied from tracheo-bronchitis to frank pneumonia, and all cases responded to treatment with antibiotics, so that no undue anxiety was caused by the respiratory disease *per se* and a virus aetiology appeared unlikely.

The explanation for these effusions remains obscure, and no patient died, except one who was readmitted 7 months after discharge and died of broncho-pneumonia and at whose autopsy no trace of the subdural effusion was found. No operations other than subdural taps were performed, and the follow-up examinations have not revealed any sequelae of local or general brain damage. All these cases occurred in Coloured* and Native children, and over the same period no European cases were seen.

Before reporting on the series of 9 cases as a whole we submit the case histories of two of the patients (cases 1 and 7).

CASE HISTORIES

Case 1. *F.S., Coloured male, aged* 5 *months*

This child was seen by one of us (W.W.) before admission. For 3 days he was feverish and restless and took his feeds poorly, and on the day before admission he developed a cough and became dyspnoeic; no convulsions, diarrhoea or vomiting. There was no history of any previous illness or of birth trauma. On examination he was found to be a well-nourished child, very ill and markedly dyspnoeic, with a temperature of 102·4°F but not dehydrated. Air entry to both lungs was good, but mild bronchospasm was present, and there were scattered rhonchi in both lung fields with crepitations over the upper zone of the right lung. There were no abnormal physical signs in the central nervous system, and the fontanelle was normal. The other systems were normal. Chest X-ray showed increased broncho-vascular markings and there were areas of patchy consolidation in the right upper zone. Further investigations are reported in Table I.

A diagnosis of broncho-pneumonia was made, and the child was treated with chloramphenicol and given oxygen and alevaire during the first day in hospital. After 48 hours he was afebrile and markedly improved generally, and the chest was clear. He continued to take his feeds poorly. On the 3rd day it was noticed that he had marked head retraction and slight tenseness of the anterior fontanelle. A lumbar puncture was done and the cerebrospinal fluid (CSF) was clear, colourless, and normal on analysis. A subdural tap was then performed and 15 c.c. of bloody xanthochromic fluid was found on the right side (Table II). After the first tap there was a marked improvement and less retraction of the head, and the child fed better.

Subdural taps were subsequently done on one or both sides at intervals ranging from 1 to 3 days, yielding 10-30 c.c. of fluid at each tap during the following 7 weeks. The quantity then diminished, and the spaces became dry after 9 weeks. A syringe was not used to withdraw fluid and surgical drainage was not carried out. At the second tap 1 c.c. of methylene blue was injected into the right subdural space and a lumbar puncture performed 4 hours later. There was no dye in the CSF, which showed that the fluid found at subdural tap did not communicate readily with the subarachnoid space. Subdural air studies and a pneumoencephalogram were done (Table II).

While in hospital he made good progress and gained 2½ lb. in weight. On discharge there were no signs of mental retardation. At follow-up 7 months later he was a healthy and normal child (Table III).

Case 7. *M.H., Coloured male, aged* 1 *year*

This child was ill for 3 days before admission, with fever and a non-productive cough, and was refusing his feeds. On the day of admission he became short of breath and irritable, and had a generalized convulsion, which lasted for a few minutes. His temperature on admission was 104°F, he was fully conscious but irritable, and dyspnoeic, and looked very ill. There was no cyanosis, and he was not dehydrated. The chest showed rib retraction at the costal margins, and over the right side in front dullness to percussion and bronchial breathing were observed. There were no adventitous sounds. In the abdomen the liver was palpable about one finger below the costal margin and the tip of the spleen could be felt. The fontanelle was open, but not bulging, and marked neck stiffness was present, with positive Kernig and Brudzinski signs. There were no other positive physical findings, and nothing significant in the past history. Chest X-ray (Fig. 1) showed dense consolidation of the entire right upper lobe. The lumbar-puncture fluid was normal. Further investigations are set out in Table I.

A diagnosis of lobar pneumonia with a febrile convulsion was made, and the child treated with penicillin and sulphadimidine. Within 24 hours the temperature came down to normal and remained so. On the 3rd day the lumbar puncture was repeated and a subdural tap performed because there was marked head retraction and the child was sucking very poorly. He had no respiratory distress at this stage, and the fontanelle was not bulging. From the left subdural space 16 c.c. of straw-coloured fluid was aspirated and 8 c.c. from the right side (Table II).

Subdural air studies were done at the time of the first tap; 10 c.c. of air was put into the left subdural space and X-rays taken (Fig. 2). Blood was taken through the anterior fontanelle from the sagittal sinus and a free flow was obtained, showing that there was no thrombosis. Subdural taps were performed

* The Coloured race is derived from a mixture of European with Malay, Hottentot, Bantu (Native) or Bushman Stock.

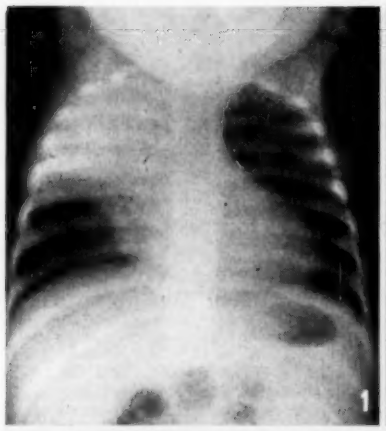

Fig. 1. Case 7. Pneumonia, right upper lobe (on admission).

Fig. 2*A*. Case 7. Left subduragram. P.A. view in upright position. Note limitation of air shadow by falx cerebri.

Fig. 2*B*. Case 7. Left subduragram. Prone position. Lateral view.

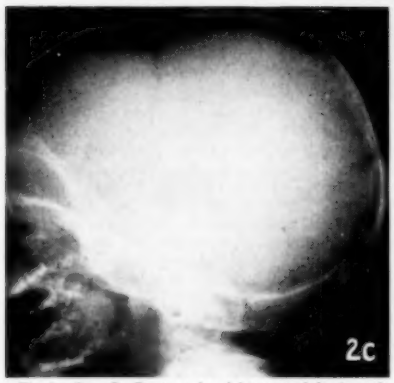

Fig. 2*C*. Case 7. Left subduragram. Upright. Lateral view.

Fig. 3*A*. Case 7. Lumbar encephalogram. P.A. upright. Normal ventricles. Air in left subdural space.

Fig. 3*B*. Case 7. Lumbar encephalogram. Lateral upright. Air present in subarachnoid and subdural spaces. Ventricles normal.

daily, and gradually diminishing amounts of fluid were obtained until on the 7th day the space was dry.

After the first tap the baby started taking the feeds well, the head retraction improved, and he gained weight during the following days.

He was discharged well on the 10th day and readmitted a week later for a lumbar encephalogram (Fig. 3) and X-ray of the chest. The chest lesion was now virtually resolved, and he had gained 1 lb. in weight since discharge. At follow-up 4½ months later, he was found to be healthy and normal, and to have gained a further 3 lb. (Table III).

STUDIES IN THE NINE CASES

Clinical Features

The clinical findings in this series are summarized in Table I. All the 9 children were admitted with chest infections, and 7 of them had radiological indications of pneumonia. The remaining 2 cases with clear X-rays had the physical signs of bronchitis and bronchiolitis.

Head retraction in all cases was the sign which drew attention to disease of the central nervous system. Only 2 cases showed abnormal fontanelles. In 6 cases the children were noted to be sucking poorly or refusing their feeds. One child had a convulsion, one vomited, and in 3 the neck was judged to be stiff. In 2 cases the children had been treated as out-patients and were admitted to hospital because of the development of head retraction.

In 5 cases subdural fluid was obtained from both sides at the initial tap, and in 2 of the remaining cases bilateral subdural effusions occurred later. In 2 cases unilateral effusions only were found. During the period under review a considerable number of cases of respiratory disease were treated as out-patients, and did not develop head retraction. Wherever this sign was found, subdural tap revealed an effusion. Usually this effusion was straw-coloured, but occasionally it was bloody. This is not to say that a subdural tap is the first examination to be made. Where signs of irritation of the central nervous system occur in the presence of infection, the first disease to be excluded must be meningitis, so that a lumbar puncture always preceded a subdural tap. In one case the CSF contained 12 cells per c.mm. and was sterile.

Following subdural taps an immediate improvement was noticed in the child's behaviour and its willingness to take feeds, and the head retraction disappeared in a few days.

The fluid obtained by subdural taps differed markedly in all cases from the fluid obtained at the same time by lumbar puncture. In 7 cases unilateral subdural air studies were made, and in no case did the air inserted cross the mid-line or fill the fissures of the brain. In case 1 a dye

TABLE I. CLINICAL FEATURES ON ADMISSION

Case Number	Age (months)	Sex	Race	Date of Admission and Discharge (1958)	Weight on Admission and Discharge (lb. and oz.)	Duration of symptoms in days	Diagnosis on Admission	Symptoms							X-ray Chest	Blood Count	Wassermann	Mantoux 1/1000	Rectal Swab	Throat Swab	Lumbar Puncture	Remarks
								Cough	Irritability	Fever	Vomiting	Diarrhoea	Convulsions	Fontanelle								
1 F.S.	5	M	Col.	21 Jul. 1 Oct.	12, 9 14, 11	3	Broncho-pneumonia	+	+	+	—	—	—	N	Patchy consol. R upper zone	Hb 10·0 g% wbc 7,800 P 57%	—	—	—	*C. albicans*	O	
2 R.Z.	3	M	Nat.	16 Aug. 1 Oct.	11, 0 11, 11	5	Lobar pneumonia	+	+	+	+	—	—	N	Consol. L lower lobe and lingula	Hb 10·5 g%	O	—	—	O	O	
3 L.W.	4	F	Col.	19 Aug. 20 Oct.	5, 11 7, 11	3	Bronchitis. Gastro-enteritis	+	—	+	+	+	—	N	NAD	Hb 11·0 g% wbc 15,600 P 38%	O	—	—	*C. albicans*	O	
4 C.H.	6	M	Col.	2 Sep. 5 Oct.	12, 5 13, 13	28	Broncho-pneumonia	+	—	—	+	—	—	N	Patchy consol. R lower lobe and L base	Hb 9·9 g% wbc 21,350 P 45%	O	—	—	O	Cells 12/c.mm Prot. 10 mg% Glob. neg. Chlor. 630 mg% Sugar N Culture sterile	Chest infection. Treated as out-patient. Head retraction.
5 S.M.	7	F	Nat.	13 Oct 8 Nov.	8, 0 8, 5	3	Lobar pneumonia	+	—	+	—	—	—	N	Consol. L upper lobe. Periosteal thickening of humeri	Hb. 9·5 g% P 40%	+	—	—	*Staph. aureus*	N	Stiff neck
6 M.B.	6	F	Col.	13 Nov. 26 Nov.	12, 8 11, 15	3	Lobar pneumonia	+	—	+	—	—	—	N	Consol. lingula	Hb 9·5 g% wbc 23,850 P 69%	O	—	—	*K. pneumoniae*	N	Stiff neck
7 M.H.	12	M	Col.	29 Nov. 9 Dec.	15, 6 16, 13	3	Lobar pneumonia	+	+	+	—	—	+	N	Consol. R upper lobe	Hb 9·6 g% wbc 8,760 P. 79%	—	—	—	*H. influenzae*	N	Convulsion
8 B.M.	5	M	Col.	12 Dec. 23 Dec.	10, 9 12, 9	28	Bronchio-litis. Gastro-enteritis	+	+	+	+	—	—	N	NAD	Hb. 12·0 g% wbc 11,000 P 58%	—	—	—	O	N	Head retraction
9 A.S.	6	M	Col.	29 Dec. 28 Feb. 59	13, 5 14, 0	14	Broncho-pneumonia. Gastro-enteritis	+	—	+	+	+	—	N	Patchy consol. R upper lobe	Hb. 11·0 g% wbc 14,500 P 68%	O	—	S. typhi-murium	*E. coli.*	O	

Col = Coloured. Nat = Native. + = present. — = absent. O = not done. N = normal. NAD = nil abnormal. Hb = haemoglobin. wbc = white cells/c.mm. P = polymorphonuclear leucocytes. R = right. L = left.

TABLE II. INVESTIGATION OF SUBDURAL AND LUMBAR FLUIDS

Case No.	History of Injury	Date of Admission (1958)	Date of First Tap (1958)	Reason	Fontenelle	Fluids at First Tap							Subdural Air Studies (1958)	Lumbar Encephalogram	Remarks
							Side	Quantity ml.	Appearance	Protein mg.%	Cells c.mm.	Culture			
1 F.S.	Nil NVD	21 Jul.	27 Jul.	Head retraction. Bulging fontanelle. Sucking poorly	Bulging	Subdural	R	15	Bloody	300	+++	S	5 Aug. L	13 Aug. Air. in L subdural space. Ventricles N	16 taps. 170 ml. in all, R 80 ml. L 90 ml.
						CSF			Clear	10	3	S			
2 R.Z.	Nil NVD	16 Aug.	25 Aug.	Head retraction. Sucking poorly	N	Subdural	R L	5 3	Bloody Straw	180	39	S	O	3 Sep. Air in both subdural spaces. Ventricles N	13 taps. 35 ml. in all, R 5 ml. L 30 ml.
						CSF			Clear	10	0	S			
3 L.W.	Nil NVD	19 Aug.	13 Sep.	Head retraction. Sucking poorly	N	Subdural	R L	3 3	Bloody Straw	100	12	S	14 Sep. L	29 Sep. Air in both subdural spaces. Ventricles N	9 taps. 23 ml. in all, R 8 ml. L 15 ml.
						C.S.F.			Clear	10	0	S			
4 C.H.	Nil NVD	2 Sep.	2 Sep.	Head retraction.	N	Subdural	R	2	Yellow			S	O	25 Sep. N	8 taps. 4 ml. in all, R 4 ml. L nil.
						C.S.F.			Clear	10	12	S			
5 S.M.	Nil NVD	13 Oct.	15 Oct.	Head retraction. Sucking poorly	N	Subdural	R	10	Yellow	50	27	S	15 Oct. R	4 Nov. N	6 taps. 42 ml. in all, R. 31 ml. L 11 ml.
						CSF			Clear	10	0	S			
6 M.B.	Nil NVD	13 Nov.	19 Nov.	Head retraction. Stiff neck. Sucking poorly	N	Subdural	R L	4 12	Bloody Yellow clot	10	3	S	19 Nov. L	25 Nov. Air in L subdural space. Ventricles N	4 taps. 19 ml. in all, R 4 ml. L 15 ml.
						CSF			Clear	10	0	S			
7 M.H.	Nil NVD	29 Nov.	2 Dec.	Head retraction. Convulsion. Stiff neck. Sucking poorly	N	Subdural	R L	8 16	Straw Straw	30	9	S	2 Dec. L	17 Dec. Air in L subdural space. Ventricles N	5 taps. 51 ml. in all, R 27 ml. L. 24 ml.
						CSF			Clear	10	3	S			
8 B.M.	Nil NVD	12 Dec.	12 Dec.	Head retraction. Stiff neck. Vomiting	N	Subdural	R L	12 1	Yellow Yellow	180	135	S	12 Dec. R	18 Dec. N	4 taps. 25 ml. in all, R 20 ml. L 5 ml.
						CSF			Clear	20	6	S			
9 A.S.	Nil NVD	29 Dec.	30 Dec.	Head retraction. Bulging fontanelle	Bulging	Subdural	L	20	Straw	40	+	S	2 Jan. 59 L	31 Jan. 59 N	12 taps. 190 ml. in all, R. nil. L 190 ml.
						CSF			Clear	20	6	S			

NVD = Normal vertex delivery. N = Normal. O = Not done. R = Right. L = Left. S = Sterile.

TABLE III. FOLLOW-UP EXAMINATION

Case No.	*Age (months)*	*Interval since discharge (months)*	*Symptoms*	*Milestones*	*Head circumference on admission and follow-up (inches)*	*Weight on discharge and follow-up (lb. and oz.)*	*Fontanelle*	*Remarks*
1	14	7	Coryza	Normal	16 18	14, 11 17, 0	N	
2	11	7	Malnutrition. Gastro-enteritis	Sitting but not crawling	16 17	11, 11 14, 7	N	Delayed milestones probably due to malnutrition.
3	12	6	None	Sitting but not crawling	— 16	7, 11 11, 13	N	Delayed milestones probably due to malnutrition.
4	14	7	Broncho-pneumonia. Malnutrition	Normal	16½ 17½	13, 13 13, 3	N	Died 4 days after 2nd admission. Autopsy—broncho-pneumonia. Nil abnormal intracranially.
5	13	5½	None	Normal	— 16¾	8, 5 14, 15	N	X-rays of bone show resolution of syphilitic process.
6	12	5	None	Normal	16 16¾	11, 15 15, 9	C	
7	16	4½	None	Normal	19 19	16, 13 19, 15	N	
8	9	4	None	Normal	— 17¾	12, 9 15, 9	N	
9	—	—	—	—	— —	14, 0 —	—	Case from afar. No reply to follow-up letter.

N=Normal. C=Closed.

was injected into the subdural space and did not appear in the CSF.

The radiological studies by lumbar encephalogram in no case showed any dilatation of the ventricular system. However, the distribution of air varied considerably and is discussed below.

Technique of Subdural Tap

Taps are usually made through the anterior fontanelle, since the posterior fontanelle is closed at birth or soon after. The scalp is shaved to expose the fontanelle and surrounding scalp, and the skin is prepared as for any surgical procedure.

It must be remembered that, once air is inserted into the subdural space, the fluid will move freely in all directions, so that meticulous positioning may be necessary. For the initial puncture it is sufficient to lay the baby on its back, suitably restrained, and insert the needle at right angles at the lateral margins of the fontanelle.

A 20-gauge lumbar-puncture needle with a short bevel should be used. After passing through the scalp a strong resistance will be felt at a depth between one-eighth and one-quarter of an inch. This is the dura mater and ommediately beneath lies the subdural space. Normally not more than a few drops of clear fluid are obtained. If no fluid is obtained the needle, with stylette reinserted, is passed forward, laterally and backwards for a short distance, as indicated by the arrows in Fig. 4. Care must be taken not to advance the needle deeply or the cortical veins may be injured. Alternatively, if possible, the needle may be inserted at right angles through the coronal suture.

In the newborn, or when the posterior fontanelle has been reopened by increased intracranial pressure, the same procedure may be carried out through the lateral margins of the posterior fontanelle to obtain fluid which has tracked backwards under the influence of gravity. A lateral insertion is essential to avoid the sagittal sinus.

After aspiration the puncture should be sealed with a sterile collodion dressing.

The aspiration should be repeated daily until less than 10 c.c. is obtained, or until the fontanelle ceases to bulge. Thereafter less frequent taps will suffice. Not more than 15 c.c. should be aspirated on each side on any one occasion.

Follow-up Studies

Details of the follow-up examination are set out in Table III. No local or general sequelae were observed during the period under review, which ranged from 4 to 7 months after discharge from hospital. One case was readmitted with broncho-pneumonia and died, and at autopsy no anatomical disorder was found within the cranium—in particular no membrane to indicate where the subdural effusion had been. It must be stated that, in this case, the amount of fluid

withdrawn was small, and subdural air studies were not done.

In 2 cases the milestones were retarded, but this appeared to be a physical rather than an intellectual defect. Coloured children are lighter in weight than their European counterparts[6] and malnutrition with delayed milestones is common.

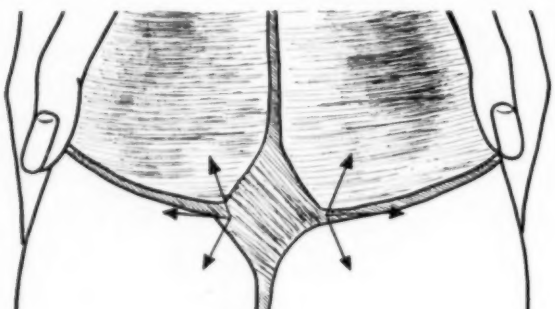

Fig. 4. Sites and directions of subdural taps (see text).

These studies do not support the plea of Williams *et al.*[1] that open operation and stripping of the subdural membrane is necessary to avoid the possibility of permanent damage. However the follow-up period is short and further studies are required.

One case of syphilis is included, and is discussed below.

DISCUSSION

The aetiology of subdural effusions has exercised the minds of many from Trotter[7] (1914) and Penfield[8] (1923) to the experiments of Finberg *et al.*[9] (1959), and the subject has been reviewed from time to time.[10-13] One supposes from reading the earlier papers that trauma due to external violence, however trivial, was considered an essential factor. It is notable that the large majority of cases occur in infants while the fontanelle is still open and before the sutures of the membranous cranium unite. It would be expected, therefore, that there would be more spring than in the older, more rigid skull, and that the release following an external impact would be more violent. For the same reason, shearing stresses would be the more easily produced. On the other hand, it is much easier to tap the subdural space at this age, and therefore disease is more likely to be discovered than later, when burr-holes have to be made.

More recently the hypothesis of internal violence has been brought forward, such as occurs with pneumoencephalography[12] and hypernatraemia.[9] In these circumstances it is suggested that the brain is stretched away from the surrounding cranium by rapid reduction in its volume, and in the process the bridging veins are torn.

In either event the principle is that a haemorrhage from veins bridging the subdural space occurs, soon followed by membrane formation and encapsulation. Fibrin and blood clot collect at the periphery of the space, and more fluid is attracted by osmosis, so that there is a tendency for the effusion to increase in size. Experimentally this has been done in animals with 'cellophane' bags[14] and with an excised subdural haematoma sac.[15] A tear in the arachnoid may also add cerebrospinal fluid.[16]

Any bleeding tendency, particularly that occurring in the first few days of life, may exaggerate any traumatic factor or cause spontaneous bleeding.

But it is not certain that haemorrhage is an essential precursor of subdural effusion, although it is difficult to postulate another cause of exudation. The anatomy of the subdural space has been compared to the synovial lining of a joint[17] in that the dura, being of mesodermal origin, has a capillary bed and is therefore capable of exudation in its own right. The inner side of the subdural space is lined by the arachnoid membrane, which is of ectodermal origin and lacks a capillary bed. Exudation can therefore only occur from the capillaries of the dura, and the arachnoid can only fill the subdural space with blood or cerebrospinal fluid.

In our series all the fluids obtained at the first tap were yellow, and of high protein content, and the majority contained no obvious blood. But since the cavity was not directly seen, it may well be that in cases where no blood was found the space was lined with blood clot and the needle withdrew fluids from the central cyst.

Had the stress of coughing been responsible for rupturing the veins bridging the subdural space, one would have expected reports of subdural effusions to be common in whooping cough, but no such reports have been seen. Our cases were not clinically dehydrated, and did not exhibit scurvy or other bleeding tendency. No history of external violence was obtained nor anything to suggest that they had not progressed mentally in a normal manner.

In case 5 the Wassermann reaction was positive and there was radiological evidence of active syphilitic bone disease. This recalls the pachymeningitis haemorrhagica interna of Virchow, originally postulated as an inflammatory cause of subdural haematoma, and now generally discarded in favour of the traumatic theory. Syphilis, then a common disease, was regarded as a cause. In this case the child was treated with penicillin and his progress has been good and healing of the bony lesions has occurred.

In case 7 a puncture of the sagittal sinus was performed at the same time as a subdural tap, and fluid blood was freely obtained, excluding a thrombophlebitis of that sinus.

Air studies were made by two methods, viz. the insertion of air direct into the subdural space by tapping, and into the subarachnoid space by lumbar puncture. The risks of the production of a subdural effusion by lumbar encephalogram are known,[13] but in all cases the presence of a subdural

effusion had been proved before the encephalogram was undertaken. The time interval between subdural and subarachnoid air studies varied from 6 to 29 days; the details are set out in Table II, and Fig. 2 is a sample. It was not uncommon to find air in the subdural space after lumbar encephalogram (cases 1, 3, 6, 7) and in these cases, except case 3, it was found on the side on which the subduragram had previously been done. It is possible, therefore, that the air had not yet been absorbed, though the interval was as long as 15 days in case 7. In case 3 only a left-sided subduragram had been done, but after encephalogram air was found in both subdural spaces. It is possible that a tear in the arachnoid, as postulated by Munro,[16] had permitted the subarachnoid air to spread to the subdural space. This view is supported by case 2, where air was found in both subdural spaces after encephalogram, though no subduragram had been done. A leakage of air at routine taps may, however, have been responsible. In cases 4, 5, 8 and 9 no air was found in the subdural spaces after encephalogram, which was done in cases 5, 8 and 9 from 6 to 29 days after subduragram (in case 4 no subduragram was done). It cannot be said, therefore, that a subarachnoid tear is present in all cases or, if it is, that it is sufficiently great to permit free passage of air between the spaces.

Since the brain trebles in size in the first year of life, any membrane adherent to its surface may interfere with the development of the underlying cortex. Williams *et al.*[4] have therefore advised that the subdural membrane should be stripped to avoid the possibility of permanent damage. We have not done this and have contented ourselves with repeated tappings in one case for as long as 9 weeks, before securing dryness.

Our patients' subsequent progress and follow-up examination have not suggested that they have been handicapped by their illness, or that they were mentally retarded children before their illness began.

SUMMARY

1. Nine cases of respiratory disease are described complicated by subdural effusions. All recovered without sequelae after antibiotic therapy and repeated subdural taps. No open operative procedures were undertaken.

2. The results of follow-up examinations are recorded.

3. No evidence of direct trauma was found in any case, and no evidence of infection of the dubdural space. One case was syphilitic.

4. No cause of this complication has been proved. There is evidence that a tear in the arachnoid may have been present in some cases. The aetiology of subdural effusions in general has been discussed.

OPSOMMING

1. Nege gevalle van respiratoriese siektes, gekompliseerd deur subdurale effusies, is beskryf. Al die pasiënte het herstel sonder gevolge na antibiotiese terapie en herhaalde subdurale aftappings. Geen ope operatiewe behandelings is gedoen nie.

2. Die resultate van opvolg ondersoeke word vermeld.

3. Geen bewys van direkte trauma of infeksie van die subdurale spasie is in enige geval gevind nie. Een van die gevalle was sifilities.

4. Geen oorsaak vir hierdie komplikasie is bewys nie. Daar is bewys dat in sommige gevalle 'n skeur in die arachnoïëd was. Die etiologie van subdurale effusies in die algemeen is bespreek.

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MODERN INSECTICIDES: THEIR TOXICITY AND CONTROL *

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Insecticides represent one sub-group of pesticides used to kill or repel insects, other pesticides being rodenticides, molluscicides, herbicides, and fumigants. These agents, some natural and many of them synthetic, generally belong to different chemical groups.

The insecticidal agents have been employed for the interior, and parts of the exterior, of dwellings and other buildings, and for the control of various insects and insect vectors of disease especially in tropical and subtropical areas. Many of these agents are toxic to man and other mammals, and their widespread use is an occupational hazard to sprayers. There is also the danger to the general population from food which may contain minute amounts of these substances. There is great hazard during their synthesis and manufacture—often far more acute than in their subsequent use, since the primary materials or processes may be more toxic than the end product.

The agents that have been used include arsenic, lead, phosphorus and thallium compounds; fluorides, cyanides, thiocyanates, nicotine, naphthalene, and numerous chlorine-containing compounds such as dicophane (DDT, chloropheno-

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thane), benzene hexachloride (BHC, lindane, gammexane), toxaphene, indane derivatives, e.g. chlordane and dieldrin; and the phosphate ester insecticides, e.g. parathion, malathion, and TEPP. A most valuable and detailed reference compendium on insecticides is available.[12]

The comparative toxicity of the insecticides for man may be arranged in the following scale. Approximate fatal doses estimated for adults are given in brackets, but it must be remembered that serious symptoms and death may occur with smaller amounts when the agents are in solution in organic solvents:

(*a*) *Extremely toxic*; fatal dose less than 1 mg. per kg. of body weight: organic phosphate esters, e.g. TEPP (10 - 20 mg.), nicotine (40 mg.), and cyanides (HCN 50 mg.).

(*b*) *Highly toxic*; fatal dose 1 - 50 mg. per kg. of body weight: Organic phosphate esters, e.g. parathion (100 mg.), arsenicals (above 120 mg. for arsenic trioxide), potassium cyanide (200 mg.), dinitroorthocresol (200 mg.) dinitrophenol (1 g.), sodium fluoride (1 g.), and naphthalene (3 g.).

(*c*) *Moderately toxic*; fatal dose 50 - 500 mg. per kg. of body weight: Pentachlorophenol (2 g.), toxaphene (2 - 7 g.), chlordane (more than 5 g.), gamma benzene hexachloride (may be 5 - 7 g., for technical product about 30 g.), and dicophane (DDT) (30 g.).

(*d*) *Slightly toxic*; fatal dose 0·5 - 5 g. per kg. of body weight: Phenothiazine (10 g.), rotenone (10 g.), derris powder (10 - 100 g.), and pyrethrum (15 — 100 g.).

CLINICAL CONSIDERATIONS AND CONTROL

Two classes of insecticides will be considered here: (A) Chlorinated hydrocarbons, and (B) organic phosphate esters.

The clinical signs of early poisoning by either the hydrocarbon or the organophosphorus compounds are not specific, and may be difficult to distinguish from an ordinary illness. The late signs of poisoning have certain characteristic features. Specific tests can establish the diagnosis of poisoning by the organophosphorus esters, e.g. depression of the blood cholinesterase, but in the chlorinated hydrocarbons it is in dicophane poisoning that a satisfactory test has been devised—the excretion of dichlorophenylacetic acid.

A. Chlorinated Hydrocarbons

1. *Dicophane (DDT, Chlorophenothane)*

This agent has been widely used as a slow-acting residual insecticide. Since certain insects, e.g. house flies and certain mosquitoes, have become resistant to this compound, other insecticides have been introduced.

Dicophane is poorly absorbed when applied to the body in solid form, but more readily when it is in solution. It is distributed in all tissues, but especially in fat, and is slowly and incompletely excreted in the urine as dichlorophenylacetic acid.

Wide individual variation in susceptibility to poisoning occurs in different animal species.

Acute poisoning in man. Dicophane is a 'cerebro-spinal poison', causing twitching of the eyelids, generalized coarse tremors and convulsive seizures, and death from respiratory failure, although heart failure due to ventricular fibrillation may also occur from sensitization of the mycoardium to hypothalamic sympathetic stimulation. In many cases the solvent has been the cause of toxic symptoms.

Treatment. The poison must be removed by gastric lavage if it has been swallowed, followed by the administration of a saline purgative and avoiding castor oil and oils or fats which promote absorption of the poison.

Phenobarbitone sodium is especially useful by subcutaneous injection, or orally, to control tremors and convulsions or other nervous symptoms, because of its selective depressant action on the motor cortex. If convulsions are severe pentobarbitone sodium is given intravenously.

Calcium gluconate injection (10 ml. of 10% solution) is given intravenously every 4–6 hours, as a nonspecific cell protectant.

Adrenaline injection must be avoided.

Control. During spraying operations with dicophane dermal exposure is much greater than respiratory exposure. A plastic cape, a hard hat with a plastic visor, and rubberized gauntlet gloves give a fully clothed man (with long-sleeved shirt and long trousers or overalls, and shoes) almost complete protection, and similar protection is afforded to other insecticides, e.g. dieldrin. For personnel working in the field, to whom the full covering with protective clothing may not be acceptable, consideration should be given to the use of a wide-brimmed, waterproof tropical helmet and plastic-netting veil with other appropriate clothing. Details are given by Wolfe *et al.*[16] The operator should stand as far as possible out of the spray drift. Hands and face should be washed before eating or smoking, and a daily bath with soap and water, and daily washing of contaminated clothing should also be strongly advised to workers. It is also important to keep the concentration of the suspension as dilute as possible.

Chronic poisoning. Repeated oral doses of dicophane were taken by volunteer men for periods up to 18 months; as much as 35 mg. daily, which is about 200 times the daily rate at which a man receives dicophane from his diet, did not produce any symptoms or signs of illness. This indicates a large safety factor with dicophane in the amounts occurring in the general diet.[13a]

2. *Gamma Benzene Hexachloride*

The gamma isomer stimulates the central nervous system.

Acute poisoning in man. Hyperirritability, muscular incoordination, clonic-tonic convulsions, and death may occur.

The delta isomer is a depressant of the central nervous system, while the alpha and beta forms produce some stimulation followed by depression. Technical benzene hexachloride is about equal to dicophane in acute toxicity to man, the gamma isomer being the most toxic fraction of the mixture; a dose of about 1 oz. of the mixture or ¼ oz. of the gamma form could be fatal to an adult.

Treatment. The measures used are the same as for dicophane poisoning. There is no specific antidote.

Control measures. See dicophane (DDT).

Chronic poisoning. The use of vaporizing and fumigating devices releasing this insecticide, for example as 'lindane', continuously day after day, has demonstrated that human beings can acquire sensitivity; angioneurotic oedema, urticaria, irritation of eyes and respiratory tract (tracheitis, asthma) have been produced, also more severe local effects, and possibly systemic effects such as polyneuritis and anaemia.[3]

3. *Toxaphene (Chlorinated Camphene)*

This potent insecticide belongs to the cyclodiene group, which also includes chlordane, dieldrin, aldrin, endrin, isodrin, heptachlor, all these being highly chlorinated cyclic hydrocarbons.

Toxaphene has a slow action, so that other agents must be used in contact sprays to ensure rapid knockdown of insects. It has been used as a substitute for dicophane.[2]

When in solution it is rapidly absorbed from the skin and other portals of entry into the body.

Acute poisoning in man. The symptoms are those of a convulsant drug affecting the brain and the spinal cord. Generalized epileptiform convulsions may occur as well as loss of consciousness and death in a few hours.

Treatment. The measures used are those described for dicophane poisoning.

Control measures. See dicophane (DDT).

4. *Chlordane*

This chlorinated hydrocarbon is a volatile liquid that has been widely used as a domestic and agricultural insecticide.[4,5] Commercial products contain a mixture of related compounds. In some fatal cases the effects have been complicated by the presence of other agents with pharmacological activity.

Chlordane is irritant to the skin (contact dermatitis) and mucous membranes. It is absorbed from the alimentary canal, respiratory tract and the skin. Preparations in the form of wettable powder have also proved toxic.

Acute poisoning in man. The symptoms are similar to those observed in poisoning by dicophane and other hydrocarbon insecticides, but are of longer duration. Irritability, salivation, laboured respiration, muscle tremors, convulsions and death may occur.

Nausea, vomiting, diarrhoea, and abdominal pain may follow the ingestion of toxic doses.

Blurred vision, cough, ataxia, confusion, delirium, and mania are further symptoms noted after inhalation and after absorption of poisonous amounts from the skin.

Symptoms may appear within 45 minutes after ingestion. Death may occur within 24 hours, frequently between 48 and 96 hours, from respiratory paralysis, but may be delayed for many days after a single oral dose. About 100 mg. per kg. of body weight of a technical mixture given orally has killed an adult.

Treatment. Poison should be removed from the skin with soap and water, or from the stomach by lavage, followed by a saline purgative, avoiding castor oil, fatty substances, and milk. A suitable barbiturate may be given, as in the treatment of dicophane poisoning.

Control measures. See dicophane (DDT).

Chronic poisoning. There may be disturbances of the central nervous system and particularly the optic nerve. Albuminuria may be produced.

5. *Dieldrin*

This agent has been used in insecticidal sprays in houses and in the field, e.g. in malaria campaigns. It is significantly more toxic than dicophane so that there is greater hazard to the operators of spray equipment. Absorption may occur from the dermal, oral, and respiratory routes. Dry dieldrin is absorbed from the skin about as easily as dieldrin in solution, in this respect differing considerably from dicophane.[15] Detailed studies of the exposure of spray-men and the hazards have been reported by Fletcher *et al.*[10,13b]

Acute poisoning in man. Giddiness and headache occur, and twitching of muscles, followed by epileptiform convulsive seizures one to several times a day or night, with loss of consciousness, lasting up to 2 hours. Mental disorder ranging from loss of memory, insomnia, and nightmares to mania have also occurred. In some cases the first convulsion occurred 15–20 days after the last exposure, perhaps due to deposition of the poison in the body and its release or activation later under conditions of stress. A recent report on poisoning was made by Patel and Rao.[14]

Treatment. Immediate washing of contaminated skin gives some protection, and other general measures are adopted. When a person has had dieldrin poisoning he should be taken off work with that insecticide, and should be examined medically for at least 2 years.

Control measures. Hands, face, and all exposed skin should be washed at frequent intervals, even though no contamination is visible, and the entire body washed with soap and water at the earliest practical moment after the day's work. Working clothes should be washed daily with soap and water. The spray-men should wear shoes and socks and a protective uniform which includes a veil of plastic netting, as described under dicophane.

B. Organic Phosphate Esters

These agents, for example parathion (thiophos) have a potent and prolonged anticholinesterase action; they are cholinergic. They are among the most toxic agents used for pest control, for instance by fruit growers and in agriculture.[1] They may be absorbed through the skin, respiratory tract, conjunctiva, and the alimentary canal. Absorption through the skin, and death, have resulted from splashes of the liquid on the skin not being removed immediately.

The symptoms are due to the inactivation of the enzyme cholinesterase, with resultant widespread effects: stimulation of the post-ganglionic parasympathetic activity, depolarization of skeletal muscle, and initial stimulation followed by depression of the central nervous system. Respiratory failure is due to broncho-constriction, neuromuscular block, and central action.

Acute poisoning in man. The principal early features may be grouped in 3 categories:

(*a*) Muscarinic symptoms: sweating, salivation, lacrimation, visual disturbances (including constricted pupils), gastro-intestinal hyperactivity, nausea, vomiting, abdominal pain.

(*b*) Nicotinic symptoms: muscle tremors in the eyelids, tongue, face and neck, followed by weakness and incoordination. The symptoms may not appear until the work is finished.

(*c*) Central nervous symptoms: early features are headache, giddiness, apprehension, and later (in severe poisoning) ataxia, confusion, speech difficulty, convulsions, severe respiratory and circulatory signs, and coma.

The acute effects last 6–30 hours, but headache, giddiness, and weakness may persist for 48–72 hours, occasionally for as long as 3 weeks after exposure. In some subjects the small pupils may not return to normal size for 3–6 weeks. The average interval between last exposure to parathion and death has been 10½ hours, and between the onset of symptoms and death 9 hours.

The diagnosis can be established by estimation of the blood cholinesterase. A convenient field kit has been devised for making the test.[9]

Treatment. The victim must be withdrawn from exposure to the insecticide. Nothing should be given by mouth to unconscious patients. Respiratory depressants should not be administered, for example morphine and barbiturates. A free airway must be maintained, and artificial respiration may be needed for many hours with administration of oxygen. Pulmonary secretions are removed by postural drainage or by suction with a catheter. Washing the skin thoroughly with soap and water, irrigation of the eyes, gastric lavage, and a saline purgative are other measures that may be indicated.

Antidotes must be given as soon as possible, e.g. (*a*) atropine sulphate, 2 mg. by intramuscular injection and repeated every 15–30 minutes, as necessary, is given to antagonize the poison, and (*b*) pyridine-2-aldoxime methiodide (2-PAM) 1–2g. (in 5% solution) is given by intravenous injection. Consciousness is restored, muscle fasciculations are stopped, and other symptoms are relieved. This drug is generally regarded as a specific anticholinesterase inhibitor, i.e. it reactivates the inhibited cholinesterase,[7,11] but this may not be the only action.[6]

Chronic poisoning. The inhibition of cholinesterase by the phosphate esters lasts 4–6 weeks, as determined by a decrease in the blood cholinesterase. Therefore, absorption of small amounts daily may eventually produce symptoms. The symptoms may then resemble those occurring in acute poisoning, but delayed weakness and paralysis of the extremities may occur (ankles and wrists) and loss of reflexes, and this may progress also to involve the respiratory muscles.

Control. Prophylactic measures must minimize absorption (*a*) from the skin—by clean protective clothing, bathing each day after work, and change to uncontaminated clothing; (*b*) from the respiratory tract—by adequate exhaust ventilation, or by wearing comfortable face-masks or respirators; (*c*) from the gastro-intestinal tract—by removal of protective clothing and thorough washing of the hands before eating, drinking, or smoking; and (*d*) by destruction or decontamination of containers and spilled material.

Strict supervision is necessary by foremen and supervisors.

Medical supervision should include routine medical examination at (weekly) intervals.

Periodic determination of the cholinesterase of the blood plasma and red blood corpuscles will help to prevent cumulative effects in those frequently exposed to these insecticides. Those with reduced cholinesterase activity should be removed from all exposure until this has returned to normal; this may take several months as the enzyme activity returns at the rate of about 1% a day.[8]

GENERAL CONTROL MEASURES TO PREVENT POISONING

1. *Education.* The dangers of poisoning by insecticides must be publicized through newspapers, magazines, films and over the radio.

Poisons should be stored in well-marked containers under lock and key. Empty containers should be burned to destroy residual poison.

2. *Stringent laws.* Proper precautionary labelling must be enforced with warnings on labels in non-fading ink (including skull and cross-bones) and information about the active ingredients, the solvent, and the antidote.

3. *Poison centres* should be established throughout the country, according to local needs and facilities in towns and cities.

4. *Cooperation of pharmacists and physicians.* At a local level they should assist in educating parents, with leaflets, pamphlets, and displays. Local doctors should be provided with information on the pharmacology of toxic agents in use and the methods used in the treatment of poisoning.

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TRANSVAAL SOCIETY OF PATHOLOGISTS

SUMMARIES OF SCIENTIFIC PAPERS *

SERUM FATTY ACIDS

A. ANTONIS, *South African Institute for Medical Research, Johannesburg*

Age, Sex and Disease Trends

Cholesteryl-ester fatty acids contain ± 50% polyenoic fatty acids, of which the major constituent is the dienoic fatty acid (linoleic acid, ± 40% of total). For White males under 30 years of age, and White females under 45 years of age, the mean value was ± 41%; with increasing age in males this decreased to ± 34%. Values for patients with coronary artery disease match those of older normals.

Triglyceride fatty acids showed a similar trend. For young males, and females, the dienoic acid content was ± 27%, decreasing with age to ± 20%. Patients with coronary artery disease have lower values (± 10-20%) than normals of similar ages; moreover their fasting serum-triglyceride levels were higher than those of normals. This suggests that patients with coronary artery disease may have an impairment in their fatty-acid transport mechanism.

Phospholipide fatty acids showed no comparable trends; the mean dienoic acid content being ± 24%, with a fairly wide scatter.

Influence of Dietary Fat

The fatty-acid composition of all classes of lipides was very responsive to dietary manipulation. Isocaloric substitution of dietary sunflower-seed oil for carbohydrate raised the proportion of linoleic acid in the triglycerides to 40-45%; in the cholesteryl-esters to 50-55%; and in the phospholipides to 30-35%. With dietary hydrogenated oil or butter, the linoleic-acid content of these lipides decreased to 15-20%, 25-30%, and 15-20%. Furthermore, on the oil diet in contrast to that of butter or hydrogenated fat, the serum total-fatty-acid levels fell significantly, paralleling similar falls in serum total-cholesterol and phospholipide levels.

* Read at a meeting of the Society held in Johannesburg on 22 November, 1958

AN ACID-FAST BACILLUS ISOLATED FROM THE LUNGS OF 'DASSIES' (*PROCAVIA CAPENSIS*)

J. C. WAGNER, *Pneumoconiosis Research Unit, Johannesburg*

An acid-fast bacillus has been isolated from granulomatous lesions found in the lungs of dassies from the New Bethesda District of the Cape Province.

This organism has similar cultural and biological features to those previously observed in the vole bacillus, *Mycobacterium muris* (Wells 1937). However, when a series of experimental animals were inoculated with similar doses of these two bacilli, the lesion caused by the vole bacillus was more severe.

THE MUTATION RATE FROM PHASE I TO PHASE II *SHIGELLA SONNEI*

J. N. COETZEE, *Institute for Pathology, Pretoria*

The morphological phase variation encountered in *Shigella sonnei* was discussed and a description was given of how the mutation rate from phase I to phase II was calculated by means of continuous culture experiments. This proved to be $6{\cdot}3 \times 10^{-4}$/bacterium/generation, which is a high rate. The back mutation rate is zero.

In vivo selective factors must operate since both phases of the organism are present in the stools of acute sonnei dysentery cases, and even carriers of the disease excrete some phase-I organisms.

A MICROMETHOD FOR THE DETERMINATION OF TRIGLYCERIDES IN BLOOD BY FLUOROMETRIC ANALYSIS

D. MENDELSOHN, *Department of Pathology and Microbiology, University of the Witwatersrand, Johannesburg*

The paucity of data concerning variations of blood triglycerides in health and disease is most probably the result of the inadequacy of current analytical methods available for triglycerides. Chromatographic techniques are rather cumbersome and time-consuming for routine use; and the chemical methods, which usually measure liberated glycerol after alkaline hydrolysis of the triglycerides, are in most cases not specific for glycerol; reproducible results are therefore difficult to obtain. The micromethod to be described is simple, specific and accurate; it utilizes the series of reactions of the Skraup quinoline synthesis. Triglycerides which have been separated from phospholipids by the two-phase extraction procedure of Delsal are hydrolysed in acid at 140°C. The liberated glycerol is subjected to the Skraup reaction with o-aminophenol in the presence of concentrated sulphuric acid and an oxidizing agent to form 8-hydroxyquinoline. The fluorescence produced by the copper chelate of 8-hydroxyquinoline in alkaline solution is then read in a Farrand fluorimeter. After separation of the triglycerides the whole reaction takes place in a single tube. The usual criteria for the validity of a method are satisfied. A straight-line graph from 0 to 500 μg triglyceride is obtained; recoveries of added triglyceride vary from 96 to 103% and the average error of the method is ± 3%. The above series of reactions has also been adapted for the estimation of free glycerol in biological fluids. Details of both procedures will be published shortly.

IDIOPATHIC AORTITIS IN BANTU CHILDREN

C. ISAACSON, *South African Institute for Medical Research, Johannesburg*

Thus far 4 autopsy and 1 possible live case of idiopathic aortitis in Bantu children have been encountered, the ages varying from 6 to 15 years. The complete autopsy findings in one case were shown, consisting of ectasia of the aorta, aneurysmal dilatation, fibrous thickening of the wall, and intimal thrombosis. One such thrombus had occluded the orifice of one renal artery, producing a Byrom-Wilson kidney. The patient in life had presented with malignant hypertension and this was the presenting feature in several of the cases. Microscopically the aorta showed a lymphocytic infiltrate that was mainly perivascular, spreading in from the adventitia to the media and intima and resulting in fibrous thickening of the adventitia and destruction of the elastic and muscle of the media with replacement, fibrosis and intimal thrombosis. The cases showed no evidence of syphilis, tuberculosis or rheumatic fever and no specific aetiological agent was identified. The histological features are similar to those described in Takayasu's disease, but also resemble the reported cases of giant-cell arteritis of the aorta, and cases described as 'idiopathic aortitis in young women'. For the moment the aetiology and histogenesis are obscure.

SOME PRACTICAL AND THEORETICAL ASPECTS OF LIVE ATTENUATED POLIOMYELITIS VACCINE

P. A. D. WINTER, *Poliomyelitis Research Foundation, Johannesburg*

Attenuated poliovirus strains with a very low degree of neurotropism, as developed by Dr. Albert Sabin of Cincinnati, USA, were studied in Dr. Sabin's laboratory for viability under field conditions of storage and for a suitable vehicle. It was found that all three types of virus were stable for at least 1 week if kept frozen, or on melting ice. At room temperature (24°C) and in the incubator (37°C) significant loss of viability occurred after 3 days.

Incorporated in an orange-flavoured glucose syrup of 25% and 60% concentration, the virus diluted to give 100,000 infective units per 4 ml. remained fully viable for 1 week when kept at 4°C. Dropped on to granulated cane sugar in a teaspoon and titrated after ½ hour the virus remained fully viable.

If the general use of such a virus can be shown to be safe as a result of numerous field trials it will be a valuable adjunct to the control of poliomyelitis. In epidemics it is rapidly effective, gives a good antibody response and, by preventing intestinal infection, should reduce the circulating virus in the population.

COLLEGE OF GENERAL PRACTITIONERS : CAPE OF GOOD HOPE FACULTY

KOLLEGE VAN ALGEMENE PRAKTISYNS : FAKULTEIT KAAP DE GOEDE HOOP

UPJOHN TRAVELLING LECTURES

The Upjohn Company has offered to finance a series of lectures to be given by teams of specialists to country practitioners, who are members of the Cape of Good Hope Faculty of the College of General Practitioners.

These lectures will take the form of week-end refresher courses in selected towns and villages in the country. The first lecture in every area will be an open lecture and a courtesy invitation will be extended to every practitioner in the district. The lectures will be arranged throughout each year and will be known as the *Upjohn Travelling Lectures*.

It is intended that the first lectures shall take place early in the New Year and suggestions and requests regarding subjects and venues will be most welcome. All those interested are invited to write to the Chairman of the Cape of Good Hope Faculty of the College of General Practitioners, Dr. F. E. Hofmeyr, 18 Worcester Road, Sea Point, Cape.

UPJOHN-REISVOORLESINGS

Die Upjohn Maatskappy het aangebied om 'n reeks voorlesings te finansier wat deur spanne spesialiste aan praktisyns op die platteland wat lede is van die Fakulteit Kaap de Goede Hoop van die Kollege van Algemene Praktisyns, gegee sal word.

Hierdie lesings sal die vorm aanneem van naweekopknappingskursusse in dorpe en dorpies op die platteland wat spesiaal vir die doel uitgekies sal word. Die eerste lesing in elke gebied sal 'n ope lesing wees en welwillendheids-uitnodigings sal aan elke praktisyn in die distrik gestuur word. Die lesings sal jaarliks dwarsdeur die jaar gereël word en sal bekend staan as die *Upjohn-reisvoorlesings*.

Dit word in die vooruitsig gestel dat die eerste lesings vroeg

in die Nuwe Jaar sal plaasvind en voorstelle en versoeke oor onderwerpe en plekke van byeenkoms sal graag ontvang word. Almal wat belangstel word uitgenooi om te skryf aan die Voorsitter van die Fakulteit Kaap de Goede Hoop van die Kollege van Algemene Praktisyns, dr. F. E. Hofmeyr, Worcesterweg 18, Seepunt, Kaap.

MEDICAL PRACTITIONERS OF UNKNOWN ADDRESS

The Registrar of the South African Medical and Dental Council has supplied the subjoined list of medical practitioners to whom communications addressed by the Registrar have been returned by the Post Office because delivery could not be effected. Attempts to trace these practitioners have failed. Their attention is invited to the following sections of the Medical, Dental and Pharmacy Act, 1928:

Section 16 (2). It shall be the duty of every registered person who changes his address to intimate the fact to the Registrar within one month after such change.

Section 17 (1). The Council may erase from the Register the name of any person who . . . (*b*) has failed, within a period of three months from the date of an enquiry sent by the Registrar by registered latter to the address appearing on the Register in respect of him, to notify the Registrar of his present address.

Section 17 (3). No person whose name has been erased in accordance with this section . . . shall be deemed to be registered unless and until on application being made by him to the Registrar, his name has been restored to the Register.

N.B. In case the practitioners concerned do not see this notice, friends who may know their present whereabouts are asked to advise them of the need to act immediately.

Aron, Pauline Lou
Bland, Campbell Young
Jones, Peter Gloag McEwan Goodman
Volker, Joachim Armin Herbert
Wannenberg, Cedric
Wittekind, Simon

IN DIE VERBYGAAN: PASSING EVENTS

Mr. Morris Brukin, M.B., B.Ch. (Rand), F.R.C.S. (Edin.), urologist, of Durban, will be available for consultations at Pietermaritzburg on Wednesdays and for emergencies at all times. His address will be 14 Braemar House, Longmarket Street, Pietermaritzburg. Telephone: 25944 Pietermaritzburg, or 24245 Durban.

Dr. Morris Brukin, M.B., B.Ch. (Rand), F.R.C.S. (Edin.), uroloog, van Durban, sal op Woensdae vir konsultasies beskikbaar wees, en ten alle tye vir noodgevalle. Sy adres sal Braemar Gebou 14, Longmarketstraat, Pietermaritzburg, wees. Telefoon: 25944 Pietermaritzburg, of 24245 Durban.

* * *

Medical Library, University of Cape Town. From 17 December 1959 until 4 January 1960 the Medical Library will not be open in the evenings. The Library will be open as usual on Monday and Thursday evenings (7.30—9.30 p.m.) from 4 January 1960.

* * *

Mr. J. Heselson, F.R.C.S. *and Dr. Sylvia Gavron* (Mrs. Heselson) left Cape Town on 30 November for an extended study tour of Europe, England and the Far East. Mr. Heselson will visit surgical centres, concentrating on peripheral vascular diseases, and Dr. Gavron will attend child guidance clinics and child psychiatric and psychological units. They will return in May 1960.

* * *

National General Practitioners' Group (M.A.S.A.). At the Annual General Meeting of this Group held at East London during the recent South African Medical Congress, the following office-bearers were elected for the ensuing 2 years: Chairman, Dr. A. G. Paterson; Vice-chairman, Dr. M. Helman; Hon. Secretary/Treasurer, Dr. Norman Levy, 210 Medical Centre, Cape Town. The Executive Committee is composed of the above elected members in addition to members of Federal Council who are general practitioners. In view of the onerous duties incumbent upon the combined post of Hon. Secretary and Treasurer, the Executive Committee has elected Dr. Alex. Gordon, Standard Bank Chambers, Muizenberg, Cape, to act as Hon. Treasurer.

* * *

International Children's Centre, Nestlé Fellowship. A fellowship of one million francs is granted every year by the French Nestlé Company to a physician who wishes to do special work in the field of nutrition. The fellowship for 1960-61 will be awarded by the International Children's Centre in the spring of 1960 (European) and candidates are requested to send the following documents to the International Children's Centre, Chateau de Longchamp, Bois de Boulogne, Paris 16e, when applying for the fellowship: (*a*) A *curriculum vitae* mentioning their work on biological and social problems concerning feeding or nutrition of infants and children, (*b*) a letter of introduction from one of their masters, and (*c*) a description of the studies which they wish to pursue with the help of the fellowship. The candidates must have a sufficient command of the French language. At the end of the fellowship the fellow, or fellows, will be expected to remit to the International Children's Centre a scientific paper on the subject which they will have studied during the year of their fellowship.

NUWE PREPARATE EN TOESTELLE: NEW PREPARATIONS AND APPLIANCES

POLYBACTRIN

Westdene Products (Pty.) Ltd. announce the introduction of a new improved container for Polybactrin, the antibiotic powder spray, manufactured by Calmic Limited of England, and supply the following information:

Widespread use of Polybactrin throughout the world has established it as a positive method of inhibiting any wound pathogens which may become implanted in the tissues during surgical procedures. Radical modification of the valve mechanism and design in the new unit give more accurate direction of the spray and wider coverage of the area under treatment. The new special valve eliminates impacting of the powder and clogging of the nozzle.

The formula and potency of Polybactrin remain unchanged—neomycin sulphate 500 mg., polymycin B sulphate 100,000 units, and zinc bacitracin 25,000 units. Recent publications[1,2] have confirmed the prophylactic value of Polybactrin in burns, general and orthopaedic surgery, gynaecological operations, etc.

Further information may be obtained from the sole South African agents, Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

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2. Turk, D. C. (1959): Canad. Med. Assoc. J., **80**, 194.

BOEKBESPREKINGS : BOOK REVIEWS

PHYSICAL DIAGNOSIS

Physical Diagnosis. 14th edition. By F. Dennette Adams, M.D. Pp. xiii+926. Illustrated. 96s. London: Baillière, Tindall & Cox Ltd. 1958.

The original author of this work was that great American teacher, Dr. Richard C. Cabot, of Harvard Medical School, who was responsible for the first 11 editions and who sought the help of the present author in the preparation of the 12th edition in 1938. Since Dr. Cabot's death the following year, Dr. Adams has taken over the work, and the extensive rewriting on account of modern developments makes it now entirely his book. It is a practical work, dealing with the conditions one is likely to meet in everyday practice, and no attempt has been made to include the unusual and the extreme. It is well illustrated, but one would have preferred certain plates in colour in addition to those of the fundi.

A.H.T.

PUBLIC HEALTH

Public Health in South Africa. 6th edition. By E. H. Cluver, K.St. J., E.D., M.A., M.D., B.Ch. (Oxon), D.P.H. (Eng.), F.R.S.I. Pp. vi + 364. 85 figures. South African price 42s. Johannesburg: Central News Agency Ltd. 1959.

On perusing the latest edition of Professor Cluver's classic work *Public Health in South Africa* we are reminded of Sir Christopher Wren's epitaph in St. Paul's Cathedral, *Si monumentum requiris, circumspice*; for this book, together with his other publications, viz. *Medical and Health Legislation in the Union of South Africa* and *Social Medicine*, constitutes the means by which he will be honoured and remembered by future generations of students. What finer monument can a writer desire? The appearance of the 6th edition will be generally welcomed by students of public health who wish to keep abreast of the great advances that have been made in this highly specialized field. Professor Cluver has dealt exhaustively with all the important aspects of public health, and he has done so in a limpid literary style which younger writers would do well to emulate. The aspects of public health covered in the book relate to the following: The human and physical environment, water, food, malnutrition, housing, refuse disposal, disinfectants, the enteric fevers, tuberculosis, leprosy, venereal diseases, plague, rheumatic fever, smallpox, poliomyelitis, influenza, rabies, trachoma, yellow fever, typhus, malaria, bilharziasis, occupational health, the pneumoconioses, cancer, vital statistics, health legislation, and cigarette smoking. All the chapters are extremely well balanced, and for students of industrial medicine those on occupational health and the pneumoconioses will prove a boon. The book is beautifully bound and excellently printed, and will bring enduring credit to the publishers as well as the author. To have it on one's shelves, is to have the vicarious pleasure of the author's companionship, which one has known across the irrecoverable years. L.F.F.

A TEXT-BOOK OF PHARMACOLOGY

Pharmacology. 5th edition. By J. H. Gaddum, M.R.C.S., L.R.C.P., Sc.D., F.R.S. Pp. xvi + 587. 93 figures. English price: 42s. London, New York, Toronto: Oxford University Press. 1959.

This text-book gives essential facts about the drugs used in therapeutics. It serves as 'an introduction to a romantic science which now has intellectual attractions, and fertile relations with the sister sciences', with practical applications in clinical medicine, veterinary medicine, and pest control. One purpose of the book is to give an account of experimental methods used in the introduction of new therapeutic agents and potentially dangerous drugs. The kind of evidence that justifies the trial of new drugs is also presented. Chemical formulae and some mathematics and other such details are also included.

The largest new sections deal with the effects of drugs on the brain and with radio-active isotopes but there are many changes throughout the book. In view of the very great increase in pharmacological knowledge this edition is 26 pages longer than the previous one and almost 25% longer than the original 1940 edition; and this only as the result of judicious pruning. All important B.P. 1958 and V.S.P. 1955 drugs are included, with doses, and also many non-official drugs. N.S.

BRIEWERUBRIEK : CORRESPONDENCE

LETTERS FROM PHARMACEUTICAL FIRMS

To the Editor: Within the past year, at least 3 letters from pharmaceutical firms have been published in the *Journal*, which comment upon articles written by medical practitioners. No reply by the author has been attached. I have been perturbed by this tendency and do not think this practice is desirable for the following reasons.

Firstly, the representatives of pharmaceutical firms can have no practical and controlled experience of the value of their products, and are dependent upon the clinical trials of others for their evaluation. Secondly, since they are marketing a product, they will always tend to look on the rosy side. Thirdly, they may stress some medically trivial aspect of a subject under review.

In my opinion the correct procedure is for the firm to direct their remarks to you, sir, for onward transmission to your contributor for reply. If you then decide to publish the correspondence it will have a more balanced appearance. In this way also any conflicting statements which the author may choose to make are put alongside those of the firm, so that both are bound to be read by interested practitioners.

Peter Suckling

406 Southern Life Buildings
St. George's Street
Cape Town
27 November 1959

POISONOUS SNAKE BITES

To the Editor: With reference to the response obtained by Dr. J. G. Botha with varidase buccal tablets in a case of snake bite[1] I should like to report a similar gratifying response in an adult who had been bitten on the left middle finger by a Bibron's adder.

There was a 'terrifying' degree of oedema and signs of haemolysis in the left arm and axilla.

In addition to other measures I prescribed twice the dosage employed in Dr. Botha's case (namely 2 tablets every 4 hours), and within 48 hours noted almost complete subsidence of the oedema.

Sidney Atlas

301 Colonial Mutual Buildings
West Street, Durban
24 November 1959

1. Correspondence (1959): S. Afr. Med. J., 33, 977 (14 November).

PAEDIATRIC NURSING

To the Editor: The Nursing Council's attention has been drawn to the Editorial 'The problem of paediatric nursing' published in the *Journal* of 7 November.[1]

The Nursing Council does not wish to comment on the Editorial as such, but must comment on the following passages in so far as the facts are concerned:

1. '2. Sisters and staff nurses of children's wards are "general-trained", i.e. their maximum average experience of paediatric nursing as trainees is 3 months.'

The period of 3 months laid down by the Nursing Council is a minimum. There is nothing which prevents training schools from posting student nurses to paediatric wards for a period of up to 5 months. The matter is entirely within the discretion of training schools.

It is interesting to note that the details of training submitted by overseas applicants often show a period of practical training in paediatric nursing of less than 3 months and not infrequently no practical training at all. Yet these persons are registered as general nurses in their countries of origin. Naturally such persons are not registered by the Nursing Council unless they undergo additional training in paediatric nursing and pass in an oral and practical examination in the subject.

2. '4. In accordance with Nursing Council requirements, it is a common practice in many hospitals for a large proportion of the nursing staff to be changed monthly. The need for adequate close supervision by properly trained senior nurses is hindered by this system. Nursing Council regulations specify a ratio of not more than 4 trainees to 1 trained nurse. Inexperienced nurses are posted into paediatric wards early in their training, before they are able to appreciate the importance of certain procedures appertaining to paediatric nursing practice.'

There is not, and never has been, a 'requirement' by the Nursing Council that the nursing staff must be changed monthly.

Neither has the Nursing Council prescribed the stage at which a student nurse shall be assigned to the paediatric wards for training; the decision rests entirely with the training schools.

3. 'If a general-trained nurse wishes to have paediatric training, she has limited opportunity for further study.'

The regulations for the training and examination for the certificate in paediatric nursing were published on 15 August 1952. To date only the Children's Memorial Hospital, Johannesburg, has applied for and is approved of as a training school. Fifty two persons have already qualified and 10 are at present in training. One wonders why, for instance, a hospital like the Red Cross War Memorial Children's Hospital has not applied for recognition as a training school.

4. 'It would appear that as from 1960 no assistant nurses, certificated or uncertificated, will be allowed to undertake nursing duties of any kind unless registered with the Nursing Council.'

It is not known what is meant by a 'certificated' assistant nurse, but attention is drawn to paragraph (*a*) of sub-section (9) of Section 12 of the Nursing Act (Act No. 69 of 1957) which reads:

'Any person who practices for gain as a nurse or midwife if he is qualified to be registered or enrolled but is not registered or enrolled shall be guilty of an offence.'

A person who is not qualified to be registered as a nurse or as a midwife or to be enrolled as an auxiliary nurse or as an auxiliary midwife may therefore practice for gain, except in prescribed areas proclaimed under section 45 of the Act.

The question whether the practice of nursing and midwifery for gain should not, in the public interest, be limited to registered and enrolled persons, is another matter.

It is, of course, within the powers of a Provincial Administration or other employing body to employ only registered and enrolled persons.

5. 'In the meantime the allocation of student nurses to the Hospital continues to fall steadily—14 for September 1959—'

It should have been made clear that the Nursing Council is not responsible for the allocation of student nurses to a hospital, nor for the recruitment of students, nor for the staffing of hospitals.

It is regrettable that incorrect facts were published in an Editorial in a publication of the standing of the *Journal*, especially in view of the alarmist publicity the matter has received in the lay press in the Transvaal (and possibly also in other centres).

C. A. Nothard
President
The South African Nursing Council
P.O. Box 1123, Pretoria
17 November 1959

1. Editorial (1959): S. Afr. Med. J., 33, 937 (7 November).

[The Editorial was a factual statement and made no attempt to allocate blame for the situation, which regrettably exists.

1. Even though the comment regarding the duration of paediatric training in the general nursing training is possibly correct, the training schools do not, in fact, usually find it expedient or possible to give 5 month's of paediatric training. The remarks on overseas nurses in this matter are irrelevant.

2. It was not alleged that the Nursing Council insisted on changing duties every month, but that to fit in with the general requirements many hospitals do so. The same applies to the stage of training. Neither custom is officially required, but both are commonly followed.

3. The number of persons trained since the publication of the relevant regulations (7 per year!) does not seem to confound the Editorial comment on the opportunities for paediatric nursing education.

4. There was indeed a terminological inexactitude, where certificated nursing assistants were referred to as certificated nurses, but the context should have prevented any misconceptions and the basic facts were not distorted thereby. It is a little difficult, however, to visualize the extension of such training by non-existent paediatric trained staff, even in the Red Cross War Memorial Children's Hospital. The application of the 1957 Nursing Act to the problem of the total inadequacy of paediatric nursing is obscure and throws no light whatever on the solution of the problem, which remains vital and urgent.

5. The remarks on the responsibility for the allocation of student nurses to a hospital are irrelevant.

May we repeat the plea made in the Editorial that this important problem be approached on a cooperative and imaginative level.—*Editor.*]

FRACTURES OF THE MANDIBLE

To the Editor: Since I have treated or been associated with at least 5,000 fractures of the facial bones over the past 22 years, I was particularly interested in the article on the treatment of fractures of the mandible which appeared in the *Journal* of 21 November.[1]

I envy the surgeons concerned their easy success in their treatment of lower jaw fractures. We have a special centre here in Pretoria, where we treat an average of 30 fractured mandibles a month. We draw our patients from the whole of the Northern and Eastern Transvaal and are continually receiving cases where complications have arisen due to inadequate treatment.

The teachers of maxillo-facial and oral surgery in this country, and abroad as well, preach that external fixation is mostly a method for aligning a fracture, but it is not a complete form of immobilization. We seldom depend on external splintage alone but also hold the teeth in occlusion by intermaxillary fixation. When there are no teeth present we also do not depend on external splintage alone, but immobilize the jaws by placing Gunning splints in the mouth as well.

With fractures at the angle, the external splint is used to hold the posterior fragment in position, but the jaws are always immobilized as well—but not for the full 5 or 6 weeks.

Even when we do an open reduction with 'figure-of-eight' wiring at the inferior border of the mandible, we do not depend on that fixation alone, but do intermaxillary immobilization for 3 weeks.

The reason for using 'braces and belt' is that we have seen too many fractures go wrong with external fixation alone and we have long since discarded reliance on this method exclusively.

The powerful muscles of the jaw set up torques, stresses and strains, which, in many cases, lead to loosening of the pins, etc. Eventually there is malocclusion of the teeth and many cases have fibrous union for life.

Malocclusion is not acceptable as an end result of treatment. Non-Europeans are notoriously long-suffering and non-complaining and they may accept results far from the ideal.

We also differ as regards non-treatment of crack fractures. We see many cases with severe complications, because someone has not bothered to immobilize a crack fracture, especially in the region of the angle of the mandible and especially when an impacted wisdom tooth is present. Infection from the mouth is pumped into the crack with every movement of the jaw, especially in the unhygienic mouth of the average non-European, and we believe that nearly always teeth in line of fracture should be removed. These teeth are often non-vital from the blow and contribute to keeping up infection.

Insufficient immobilization and lack of removal of teeth in line of fracture are the most frequent cause of the complications we see.

I could reply at length to the discussion covering the drawbacks of intermaxillary fixation, but I will be brief. I cannot remember ever removing teeth to allow feeding and have seldom been confronted with excessive loss of weight. We have no difficulty in removing wires and always open up the mouth for the anaesthetists.

I believe that every large hospital ought to have the services of a specialist maxillo-facial and oral surgeon. There are a number being trained in South Africa at the moment and they will soon be available.

Finally, I must congratulate the surgeons of Edenvale Hospital in getting as good results as they do in the face of lack of specialist facilities.

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